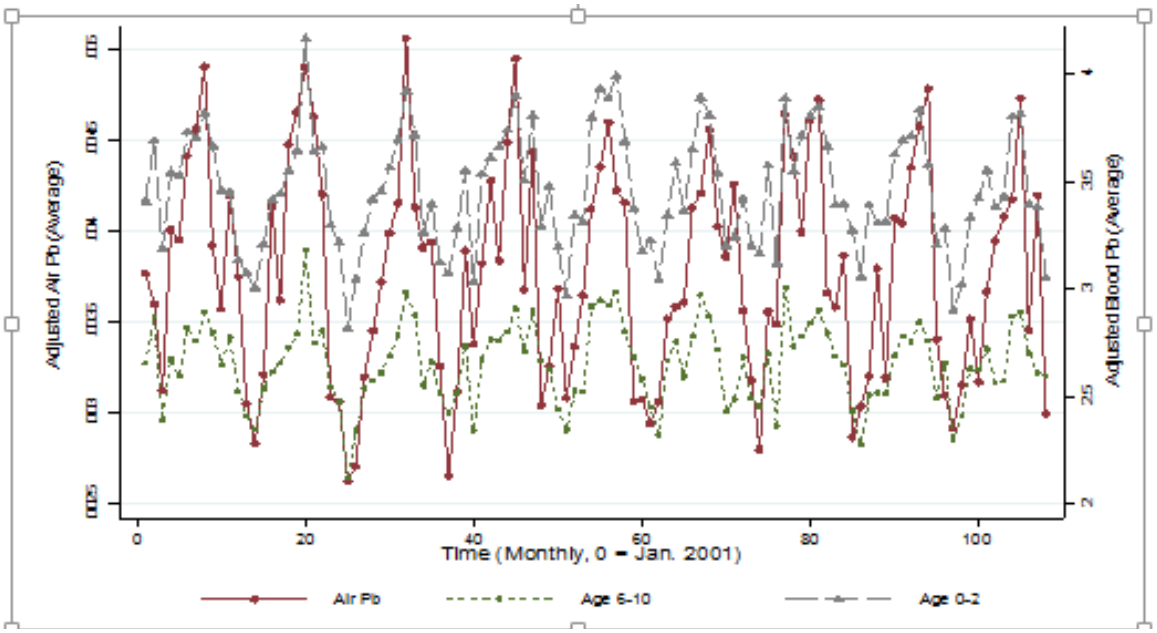
Soil lead and human health exposure risks: Studies from Australia and the United States of America



Weather-adjusted air lead (Pb) (µg/m^3) and Blood Pb (PbB) (µg/dL) by age group in Detroit, Michigan. Average monthly child PbB levels adjusted by local weather conditions, child gender, method of blood draw, and census tract fixed effects. (Source = Zahran et al., 2013a)

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ABSTRACT

Urban surface soils have been contaminated with lead (Pb) primarily from the former use of Pb additives in petrol and Pb paint, and in some instances from Pb smelters. These exposures continue to pose an ongoing risk to human health globally. Lead is a neurotoxin. When it is absorbed, inhaled or ingested, it can affect the development of the child's nervous system causing lower intelligence quotient measures, Attention Deficit Hyperactivity Disorder (ADHD) and delinquent behaviors.

In a series of 8 published peer-reviewed papers (and one response to comments paper), this thesis assesses soil Pb contributions to blood Pb (PbB) in Australia and the USA. In addition, the study assesses the role of Pb additives in petrol (gasoline) as a potential source of blood PbB in children. In evaluating the potential role of petrol Pb additives for elevating children's PbB levels and urban soil Pb levels, the spatial and temporal variation of Pb in atmospheric and household dusts were evaluated.

The results from the thesis studies demonstrate that the historical use of leaded gasoline and Pb in exterior paints has contaminated urban soils to levels that pose a potential risk of harm to children. Leaded gasoline is a major source of Pb urban soils and house dust. Children's PbB levels are associated spatially with soil Pb concentrations and temporally with atmospheric soil and Pb concentrations. Roadside soils contaminated with Pb are subject to re-suspension by vehicle movement, which causes dispersal into the urban environment.

This thesis indicates that the paradigm that Pb paint is *the* sole primary source of Pb exposure in urban children is incorrect. Ongoing exposure from legacy deposition of Pb from petrol is also a major source of exposure in children and still poses a significant risk of harm.

ACKNOWLEDGEMENTS

Several people and organizations have helped me with this project and I would like to take this opportunity to thank them.

My highest appreciation goes to my wife Lisa Hayden and daughter Isobella Laidlaw who came to Australia to allow me to pursue a PhD and stayed with me in Australia for eight more years. Lisa's assistance was crucial as she encouraged me to keep going when I was about to quit. My Father (deceased) and Mother greatly contributed by supporting my move to Australia to pursue a PhD and gave abundant support while I was in Australia. My sisters Kari Laidlaw and Heather Schneider also supported me emotionally when I was isolated in a foreign land. Professor Mark Taylor was very flexible and very inspirational and gave me the freedom to pursue my own study design and encouraged my collaboration with other colleagues. He has been a great mentor. I greatly appreciate Macquarie University's support for funding me. A very special thank you goes to the 5 Sydney Residents who gave me a great deal of their time and put up with monthly inconveniences for 15 months – this study would not have been possible without them. I gratefully acknowledge Emeritus Professor Brian Gulson and Karen Mizon for their assistance at the start of my thesis in introducing me to two Sydney residents involved in the Sydney study. I also thank Associate Professor Damian Gore, Macquarie University for his useful occasional advice on technical aspects of my PhD. Russell Field was very helpful and assisted with my laboratory studies. Glyn Devlin and the Australian Synchrotron deserve special thanks for providing funding and access to the Synchrotron. I thank ALS Laboratories for their metal analysis and the National Measurement Institute for their analysis work on Pb isotopes. These high quality analyses were critical to my 5 home Sydney study. I am indebted to Professor Gabriel Filippelli who continued to collaborate with me when I was no longer enrolled at IUPUI and who has put up

with my obsession with soil and lead poisoning for a long time. I am also extremely grateful to Professor Howard Mielke who agreed to collaborate with an unknown and unpublished student (me) after we met in Scotland in 2003. He deserves the Nobel Prize for his pioneering work in this field. I have been very fortunate to meet the super skilled Assistant Professor Sammy Zahran who has contributed greatly to our papers. I have also been very lucky to work with Professor Nicholas Pingitore and Dr. Juan Clague of the University of Texas at El Paso who have graciously assisted with the interpretation of the synchrotron data. I am very thankful that Assistant Professor Shawn P. McElmurry was willing to collaborate and play a crucial role by obtaining the Detroit child PbB database. Dr. Charles Ritter deserves a special recognition for stimulating my interest in heavy metals in soils and the atmosphere while I was an undergraduate at the University of Dayton, way back in 1993. Without his influence, this thesis would not have been pursued. Professor Don Pair and Mr. George Springer (and others) were also very inspirational at the University of Dayton geology department. Linda Patrick's editing assistance was also invaluable.

This dissertation is dedicated to my father Duncan M. Laidlaw (deceased) who was very kind throughout the years and contributed to my interest in science through his passion for plants and flower gardens.

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STATEMENT OF CANDIDATE

I certify that the work in this thesis entitled "*Soil lead and human health exposure risks: Studies from Australia and the United States of America*" has not been submitted previously, in whole or in part, for a degree at this or any other university. The thesis does not contain, to the best of my knowledge and belief, any material published or written by another person, except where acknowledged. I certify that this thesis is an original piece of research that is comprised of solely my own work.

Mark Andrew Scott Laidlaw, 1 April, 2014

NOTES REGARDING THESIS FORMATTING AND STATEMENT OF CONTRIBUTION

This thesis is structured into six chapters and two appendices. The first chapter consists of a broad-scale introduction to the research principles and study area. The underlying research aims and objectives are addressed over eight published papers (and one response to comments paper) that make up Chapters 2 to 4, introduced below. The thesis chapters are comprised of individual journal articles or groups of individual journal articles that contribute towards answering the research questions and aims of the thesis. Chapter 5 consists of the Discussion and Chapter 6 the Conclusions.

Chapter 2: Petrol derived lead (Pb) in urban surface soils is a major source of Pb in house dust and has the potential to poison children in the inner cities of Australia.

1) **Laidlaw MAS** (75%), Taylor MP (25%). 2011. Potential for childhood Pb poisoning in the inner cities of Australia due to exposure to Pb in soil dust. Environmental Pollution 159(1), 1-9.

   This paper was largely my own conception, development and execution with direction from Mark Taylor.

2) **Laidlaw M.A.S. (**60%), Zahran S. (10%), Pingitore N. (2%), Clague J. (2%), Devlin G. (1%), Taylor M.P. (25%). 2014a. Identification of lead sources in residential environments: Sydney, Australia. Environmental Pollution. (Accepted).

   This original idea for this paper was my own and I directed its development, the text and its execution. I performed the field-work. Taylor assisted with some

aspects of the study design, fieldwork, mentoring, development of text, reviewing and final drafting of the manuscript. Zahran performed advanced statistical analysis and wrote some of the results section, Clague extracted the synchrotron data and plotted the data into charts, Pingitore wrote the results section for the synchrotron samples and Devlin assisted with the synchrotron sample analysis and wrote the synchrotron methods section.

3) **Laidlaw M.A.S.** (60%), Zahran S. (5%), Pingitore N. (5%), Clague J. (0%), Devlin G. (0%), Taylor M.P. (30%). 2014b. Response to comments on: Identification of lead sources in residential environments: Sydney Australia. By Laidlaw, M.A.S., Zahran, S., Pingitore, N., Clague, J., Devlin, G., Taylor, M.P., 2014. Environmental Pollution 184, 238-246

   The Laidlaw et al. (2014b) paper contains Laidlaw et al.'s response to comments made by Brian Gulson (Gulson, 2014) about the Laidlaw et al. (2014a) paper.

Chapter 3: Calculation of historical vehicle traffic Pb emissions in US and California urbanized areas and its legacy in urban soils and continued effect on children's health.

1) Mielke H.W. (55%), Laidlaw M.A.S. (40%), Gonzales C.R. (5%). 2011. Estimation of leaded gasoline's continuing material and health impacts on 90 US urbanized areas. Environment International 37(1), 248-57.

   While this paper was largely Howard Mielke's conception, development and execution, I developed approximately 40% of the text and compiled and analysed the USA literature on soil Pb studies.

2) Mielke H.W. (55%), **Laidlaw M.A.S.** (30%), Gonzales C.R. (5%). 2010. Lead (Pb) legacy from vehicle traffic in eight California urbanized areas: continuing influence of Pb dust on children's health. Science of the Total Environment 408(19), 3965-75.

Although this paper was largely Howard Mielke's conception, development and execution, I developed around 30% of the text, including the section on analysis and discussion.

Chapter 4: Soil Pb and children's blood Pb (PbB) levels are associated spatially and temporally in urban areas.

1) **Laidlaw M.A.S.** (65%), Zahran S. (20%), Mielke H.W. (5%), Taylor M.P. (5%), Filippelli G.M. (5%). 2012. Re-suspension of Pb contaminated urban soil as a dominant source of atmospheric Pb in Birmingham, Chicago, Detroit and Pittsburgh, USA. Atmospheric Environment 49, 302-310.

I had the original idea for this manuscript in 2009 when I published the following conference paper: *Laidlaw, M.A.S. 2009. Correlation of Atmospheric Soil and Atmospheric Pb in Three North American Cities: Can Re-suspension of Urban Pb Contaminated Soil be a Major Source of Urban Atmospheric Pb and Cause Seasonal Variations in Children's PbB Levels? 24th International Applied Geochemistry Symposium. New Brunswick, Canada.* I further developed this pilot study by adding a fourth city. Sammy Zahran took the natural logs of the soil and atmospheric data and discovered the significant difference in the weekly versus weekend values. We hypothesised this was due to changes in traffic volumes and

its effect on contaminated dust re-suspension. Mielke, Filippelli and Taylor assisted with editing.

2) Zahran S. (40%), **Laidlaw M.A.S.** (30%), McElmurry, S. (20%), Taylor M. (5%), Filippelli G.M. (5%). 2013a. Linking Source and Effect: Re-suspended Soil Pb, Air Pb, and Children's PbB Levels in Detroit, Michigan. Environmental Science and Technology 47(6), 2839-45.

The original idea for this manuscript was mine. I identified seasonal PbB curves for Detroit from web published data (Shawn McElmurry) for the period 2001 to 2009. I then extracted seasonal atmospheric soil and Pb data for one of the Detroit IMPROVE stations located on the IMPROVE database (IMPROVE, 2013) and compared the datasets where I observed clearly that the PbB and Child PbB peaks coincided in the summer and autumn time. I then contacted Shawn McElmurry and asked if he could obtain the Detroit child PbB database for comparison to the IMPROVE air Pb data. I asked Sammy Zahran to assist with the complex statistical analysis of the data relationships. Gabriel Filippelli assisted with editing. Mark Taylor assisted with the formulation of the argument, data interpretation, manuscript editing and writing.

3) Zahran S. (50%), Mielke H.W. (20%), Filippelli G.M. (7.5%), McElmurry S. P. (7.5%), **Laidlaw M.A.S.** (7.5%), Taylor M.P. (7.5%). 2013b. Determining the relative importance of soil sample locations to predict risk of children's lead (Pb) exposure. Environment International 60, 7-14

The concept for this study was developed by Sammy Zahran, Howard Mielke and myself while much of the development and execution were performed by Zahran and Mielke. My main contribution was to identify and argue that the best contribution of this paper was its potential future use by other researchers to select soil sample locations in an urban area in a manner which limits the number of samples required to assess spatial patterns of children's PbB levels. This assisted with the overall arrangement of the paper. I also performed editing and writing of some sections of the work. Taylor, McElmurry and Filippelli performed writing and editing.

4) Filippelli, G.M. (90%) and **Laidlaw,** M.A.S. (10%). 2010. The Elephant in the Playground: Confronting Pb-contaminated soils as an important source of Pb burdens to urban populations. Perspectives in Biology and Medicine 53, 31-45.

This paper was largely Gabriel Filippelli's conception, development and execution. I developed approximately 20% of the text and performed editing and proofing.

CHAPTER 1

Introduction, aims and approach to the study

1.1 GENERAL INTRODUCTION

In the 1950s, Cal Tech geochemist Clair Patterson was conducting experiments to pinpoint the age of various rocks, but noticed repeatedly that his results were skewed consistently as a result of sample lead (Pb) contamination. This finding resulted in Patterson seeking to understand what the source and cause of this persistent contamination was. Further studies showed that Pb levels were elevated in certain waters, soils, organisms (Settle and Patterson 1980), even Arctic ice—and most troubling, in the human body. Over the next three decades, Patterson helped to promote a crusade against the use of Pb that attracted the vociferous opposition of industry groups. Professor Patterson eventually convinced lawmakers and regulators to outlaw Pb in pipes, solder, and finally in petrol (gasoline) (Bryson 2003). Professor Clair Patterson stated the following in 1980: "*Sometime in the near future it probably will be shown that the older urban areas of the United States have been rendered more or less uninhabitable by the millions of tons of poisonous industrial Pb residues that have accumulated in cities during the past century……Extrapolating from present information, ...probably... it will be shown in the future that average American adults experience a variety of significant physiological and intellectual dysfunctions caused by long-term chronic lead insult to their bodies and minds which results from excess exposures to industrial lead that are five hundred-fold above natural levels of lead exposure, and that such dysfunctions on this massive scale may have significantly influenced the course of American history.*" (NRC, 1980). This pivotal and controversial statement of Clair Patterson's provided the basis for this PhD inquiry.

Blood lead (Pb) studies show that urban children in the United States (and potentially Australia) have disproportionately elevated PbB levels relative to their suburban and rural counterparts (Filippelli et al., 2005). However, the scientific, academic and practical opinions

and responses as to the source and cause of Pb that is poisoning children are often polarized into two ‘camps’. One ‘camp’ argues that Pb paint is the main source of exposure (e.g. Jacobs, 1995; Brown and Jacobs, 2006) while another ‘camp’ argues that Pb from soil, which has primarily been contaminated from the past use of Pb as an additive in petrol, is the main source (e.g. Mielke and Reagan, 1998; Mielke, 1999). In some ways this polarization has been problematic because it has stymied the necessary focus of remediation efforts in contaminated areas.

In New Orleans, Louisiana, Mielke et al. (1997) first observed on a large scale that soil Pb was spatially associated with children’s PbB levels. In addition, for many years it has been observed that the dominant temporal maxima in urban children’s PbB levels in the United States occur during the summertime and autumn with lower values being recorded in the winter and spring seasons (USEPA, 1995; USEPA, 1996). Child PbB seasonality has been observed in at least 12 locations in the United States (see Section 1.13). Until recently, the cause of these temporal trends in Pb levels has not been well articulated (Laidlaw et al., 2005; Laidlaw and Filippelli, 2008). Understanding this temporal pattern could indicate a major source of Pb in urban children and point the way to effective remedial efforts. Australia does not collect systematic PbB data from children, and consequently it is not known if seasonal PbB patterns exist in this country. In limited data sets (n = 7 children - Gulson et al., 2000; n = 37 children - Gulson et al., 2008) from the Sydney area, Gulson et al., (2000; 2008) reported that PbB seasonality was not observed in Sydney children.

Prior to the publication of the papers in this dissertation, only two papers displayed associations between children’s PbB seasonality and *external* environmental variables. Laidlaw et al. (2005) was able to predict, using regression modelling, children’s seasonal PbB

variation in Indianapolis, Syracuse and New Orleans through the use of independent variables such as wind speed, temperature, soil moisture and particulate matter less than 10 microns (sometimes expressed as PM_{10}) – variables used to predict soil re-suspension (Edfelson and Anderson, 1943; Cornelis and Gabriels, 2003). In Milwaukee, Wisconsin, Havlena et al. (2009) observed that seasonal variations in particulate matter less than 2.5 microns ($PM_{2.5}$) were correlated with 10 month old children's seasonal PbB levels. Other than these studies, no other papers historically were able to successfully model the seasonal variations in children's PbB levels using atmospheric Pb concentrations. The limitation of the Laidlaw et al., (2005) and the Havlena et al. (2009) papers was that they indirectly predicted children's PbB levels, rather than directly predicting children's PbB levels using atmospheric Pb levels. Atmospheric Pb data was either not available or not used when these studies were completed.

The principal aim of this thesis is to demonstrate that *the* major source of high prevalence *low-level* Pb poisoning (Zahran et al., 2013a; 2013b) in urban children is Pb in soil-derived dust, and that the principal source of Pb in this dust is from vehicle emissions during the period when Pb was used in petrol. The prevailing paradigm is that Pb paint is the source of this poisoning. Pb paint is actually associated with the lower prevalence of moderate to high PbB levels in urban children (McElvaine, 1992). It is acknowledged that the PbB source apportionment issue is very complex and the source of Pb in children's blood varies and can change depending on the place and time and PbB concentration. Indeed, studies have shown that Pb in children's blood may originate from a multitude of sources: legacy petrol-derived Pb in urban soil dust reservoirs (Mielke and Reagan, 1998), paint (Rabinowitz, 1987) and paint chips (McElvaine et al. 1992), Pb water lines (Edwards et al., 2009), combustion of municipal solid waste (Chillrud et al., 1999) or general aviation airplane fuel (avgas) (Miranda, 2011) and other minor sources.

1.2 LEAD TOXICITY

The current goal for all Australians is a PbB concentration of < 10 µg/dL (NHMRC, 2009). However, emerging evidence (see below) suggests that the definition of Pb poisoning in Australia may need to be reduced to 5 µg/dL, or even lower at 2 µg/dL (Taylor et al., 2010b). In Australia, this change could result in the emergence of a large number of children being defined as Pb poisoned. A recent WHO Childhood Lead Poisoning Report (WHO, 2010; p. 12) makes the following comments with respect to the adverse effects of Pb exposure: "*Recent research indicates that lead is associated with neurobehavioural damage at blood levels of 5 µg/dl and even lower. There appears to be no threshold level below which lead causes no injury to the developing human brain*." Low PbB levels (<10 µg/dL) typically associated with urban soil Pb exposure are associated with a myriad of adverse health outcomes. Canfield et al. (2003) observed that when lifetime average PbB concentrations in children increased from 1 to 10 µg/dL, the intelligence quotient (IQ) declined by 7.4 points. Jusko et al. (2008) observed that compared with children who had lifetime average PbB concentrations < 5 µg/dL, children with lifetime average concentrations between 5 and 9.9 µg/dL scored 4.9 points lower on Full-Scale IQ (91.3 vs. 86.4, p=0.03). Similarly, Surkan et al. (2007) observed that children with 5-10 µg/dL had 5.0 (S.D. 2.3) points lower IQ scores compared to children with PbB levels of 1-2 µg/dL (p = 0.03). Interestingly, multiple studies have shown that the strongest Pb effects on IQ occurred within the first few micrograms of PbB (Canfield et al., 2003; Lanphear et al., 2005; Schnaas et al., 2006). Low PbB levels (<10 µg/dL) have also been associated with various physiological outcomes such as kidney damage (Fadrowski et al., 2010), dental caries (Moss et al., 1999), puberty delay in boys (Williams et al., 2010) and girls (Selevan et al., 2003) and cardiovascular outcomes in adults

(Navas-Acien et al., 2007).

Low level Pb exposure is of great concern as some researchers now conclude that Pb is no longer considered safe at any dose (Bellinger, 2011). Bellinger (2011) reviewed the health effects associated with Pb and found associations between Pb and mortality, renal disease, cardiovascular disease, impaired reproductive outcomes, nerve conduction velocity reductions, postural balance issues, essential tremors, amyotrophic lateral sclerosis, adult cognitive function, adult psychiatric status, brain white matter lesions, children's IQ and neuropsychological function, children's attention deficit hyperactivity disorder (ADHD) and dental health.

1.3 BLOOD LEAD AND VIOLENCE

A new research paradigm is emerging in regard to the toxicity effects of Pb exposure and its association with violence and delinquent behaviour. In an early study, Needleman et al. (2002) observed that bone Pb levels were associated with delinquent acts. Dietrich et al. (2001) performed a prospective longitudinal birth cohort of 195 urban, inner-city adolescents recruited between 1979 and 1985. Relationships between prenatal and postnatal exposure to Pb (serial PbB determinations) and antisocial and delinquent behaviours (self- and parental reports) were examined. Prenatal exposure to Pb was significantly associated with a covariate-adjusted increase in the frequency of parent-reported delinquent and antisocial behaviours, while prenatal and postnatal exposure to Pb was significantly associated with a covariate-adjusted increase in frequency of self-reported delinquent and antisocial behaviours. Nevin (2007) showed a very strong association between preschool PbB and subsequent crime rate trends over several decades in the USA, Britain, Canada, France, Finland, Italy, West Germany, and New Zealand. Wright et al. (2008) performed a study in

Cincinnati, Ohio and concluded that prenatal and postnatal PbB concentrations are associated with higher rates of total arrests and/or arrests for offenses involving violence. Carpenter and Nevin (2010) indicate that recent evidence suggests that temporal trends in rates of violent crime in many nations are consistent with earlier preschool PbB trends, with a lag of about 20 years. These associations are consistent with many controlled studies suggesting that Pb-exposed children suffer irreversible brain alterations that make them more likely to commit violent crimes as young adults. Mielke and Zahran (2012) evaluated air Pb emissions and latent aggravated assault behaviour in Chicago, Illinois, Indianapolis, Indiana Minneapolis, Minnesota San Diego, California Atlanta, Georgia and New Orleans, Louisiana. Other things held equal, a 1% increase in tonnages of air Pb released 22 years prior raises the present period aggravated assault rate by 0.46% (95% CI, 0.28 to 0.64). Overall their model explains 90% of the variation in aggravated assault across the cities examined. In the case of New Orleans, 85% of temporal variation in the aggravated assault rate is explained by the annual rise and fall of air Pb (total = 10,179 metric tons) released on the population of New Orleans 22 years earlier. Nriagu (2011) reviewed recent studies demonstrating a link between Pb poisoning and violence and delinquent behaviour. Similarly, in Australia, Taylor et al. (2013), used data from the NSW Bureau of Crime Statistics and Research and the NSW Environment Protection Authority (EPA) and examined the correlations between Pb-in-air emissions and crime rates with 20- and 21-year time lags at seven sites in NSW. It was observed that all seven sites showed that higher levels of airborne Pb resulted in higher assault rates 20 to 21 years later and that areas with higher Pb levels tended to show stronger relationships.

1.4 BLOOD LEAD AND SCHOOL OUTCOMES

Lower PbB levels (<10 μg/dL) typically associated with urban soil Pb exposure are now being associated with health outcomes related to subtle nervous disorders. Needleman et al. (1979) was one of the first researchers to identify the impact of children's early Pb exposure to classroom performance. Low PbB <10 μg/dL is associated with ADHD (Nigg et al., 2010) and a reduction in children's test scores for reading (Odds Ratio (OR) 0.51, p=0.006) and writing (OR 0.49, p=0.003) and mathematics (Chandramouli et al., 2009; Miranda et al., 2007).

Zhang et al. (2013) assessed the long-term effect of early childhood Pb exposure on academic achievement in mathematics, science, and reading among elementary and junior high school children in Detroit, Michigan. They observed that high PbB levels before age 6 years were strongly associated with poor academic achievement in grades 3, 5, and 8. Reduced student performance has been demonstrated as one of the characteristics of the accumulation of Pb dust in communities of urban areas (UAs). The literature on the effects of Pb on student performance is large (Martin, 2008). Support for the idea that soil Pb in a community is associated with student performance was indicated in Minnesota by the observation that dropout rates of high school students followed city size and soil Pb (Mielke et al., 1989). In New Orleans, soil Pb on the play areas of elementary public schools follow the same trend as the urban soil Pb map of the entire city; however, soils on school grounds are significantly less Pb contaminated than soils of neighbouring residential properties where the most vulnerable preschool children reside and play before they attend school (Higgs et al., 1999). New Orleans school achievement test scores were found to be associated with soil metals including Pb (Mielke et al., 2005b; Mielke and Berry, 2007, pp 116–123). Finally, standardized test performance of children attending local schools is significantly linked with

soil Pb and PbB of the same school district. This phenomenon indicates the serious neurotoxicity effects of Pb (Zahran et al., 2009). These studies characterize the more insidious and costly burdens that environmental Pb imposes on urban society (Campanella and Mielke, 2008; Chandramouli et al., 2009; Gould, 2009; Nigg et al., 2008; Zahran et al., 2009).

1.5 URBAN LEAD POISONING EPIDEMIC

In the United States, there is an epidemic of children's Pb poisoning in many of the older inner-city areas which experienced a large volume of traffic flow between the 1920s and the 1980s (the main period of leaded petrol use) and which can also contain older homes coated with Pb-based paints. It has been estimated that 24.5%, or 9.6 million US children have a PbB in the 2–10 μg/dL range, a level which will cause clinical signs (Gould, 2009). New Orleans children currently have a PbB prevalence (> 5 μg/dL) of 29.6% (Mielke and Zahran, 2012) and Detroit currently has a PbB prevalence of 33% (> 5μg/dL) (Zahran et al., 2013a). A graphical representation of the child PbB epidemic in Detroit, Michigan and Milwaukee, Wisconsin can be observed in Figures 1.1 and 1.2 below. The PbB prevalence in Australian inner cities has not recently been determined, other than in the Donovan (1996) report and other small samples studies (Gulson et al., 2006; Gulson et al., 2008). The Donovan (1996) study is the only Australian national study to measure the PbB of children aged 1 to 4 years (n = 1,575). In a portion of homes, Pb concentrations were analysed in soil, paint, water and house dust samples. The study observed an arithmetic mean PbB level of 5.72 μg/dL and a geometric mean PbB level of 5.05 μg/dL. The PbB prevalence in Australia is not measured systematically by the state health departments except in the towns of Broken Hill, Port Pirie and Mount Isa (Taylor et al., 2011), where there are known, ongoing Pb exposure issues in children.

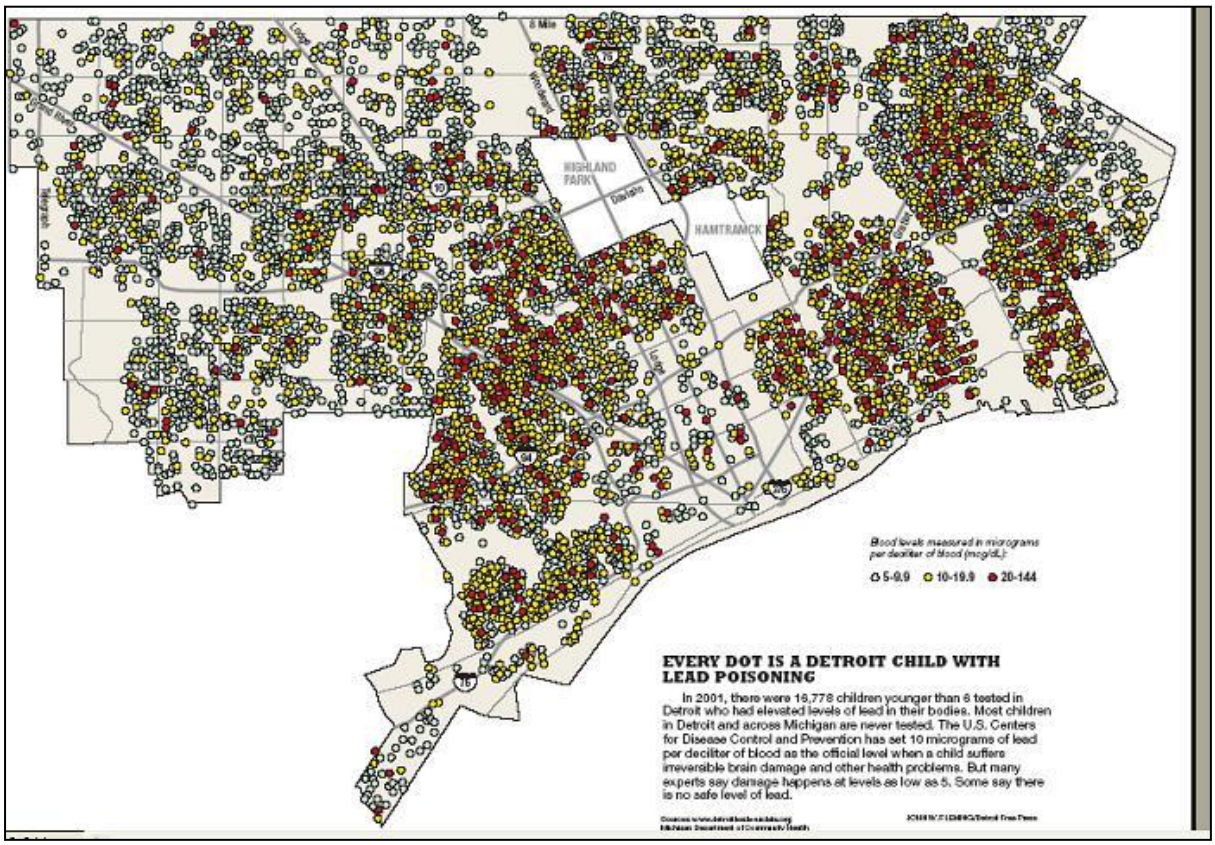

Figure 1.1 – This map contains points which represent the incidence of PbB poisoning in Detroit, Michigan in 2001 (Wayne State, 2012). White circles represent a child with a PbB level of 5 to 9.9 µg/dL, yellow circles represent a child with a PbB level of 10-19.9 µg/dL and red circles represent a child with a PbB level > 20 µg/dL.

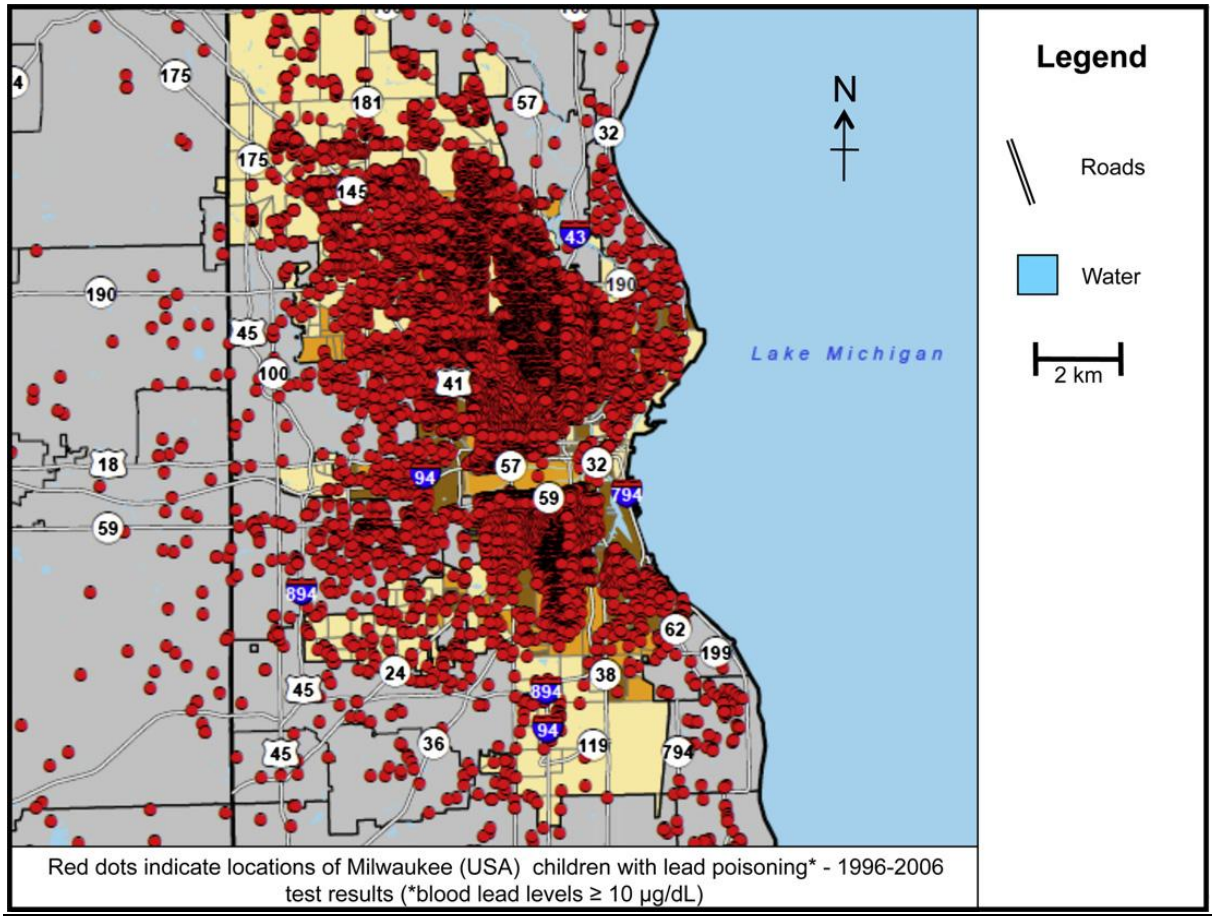

Figure 1.2 - Milwaukee, Wisconsin Pb Poisoning Map: 1996-2006. This map depicts the 36,856 children that exhibited PbB poisoning > 10 µg/dL in Milwaukee between 1996 and 2006 (source: Laidlaw and Taylor, 2011).

1.6 LEAD AS AN ADDITIVE IN PETROL IN AUSTRALIA

In Australia, leaded petrol was one of the major anthropogenic sources of Pb in the atmosphere between 1932 and 2002 (Gulson et al., 1983; Cook and Gale, 2005). It has been estimated that leaded petrol emissions contributed up to 90% of the atmospheric Pb in UAs in the country (NEPM, 2001). In two national assessments of petrol Pb emissions, it was determined that in 1976, 3,842 tonnes of Pb were emitted in Australian capital cities and 2,388 tonnes of Pb were emitted in 1985 (Farrington and Boyd, 1976; Farrington, 1985). Unleaded petrol became available in 1986, and comprised 37% of total fuel sales in 1991 (Rossi, 2008) and 50% of total fuel sales in 1993 (Bollhöfer and Rosman, 2000). In January 2002, the National Fuel Quality Standards Act 2000 (Department of the Environment and

Heritage, 2005) banned the content of Pb in petrol above 0.005 g/L. Figure 1.3 displays the number of motor vehicles in Australia between 1928 and 1991 (Cook and Gale, 2005). This figure suggests that the total automotive Pb emissions in Australia increased between 1928 and 1991 as the number of vehicles increased.

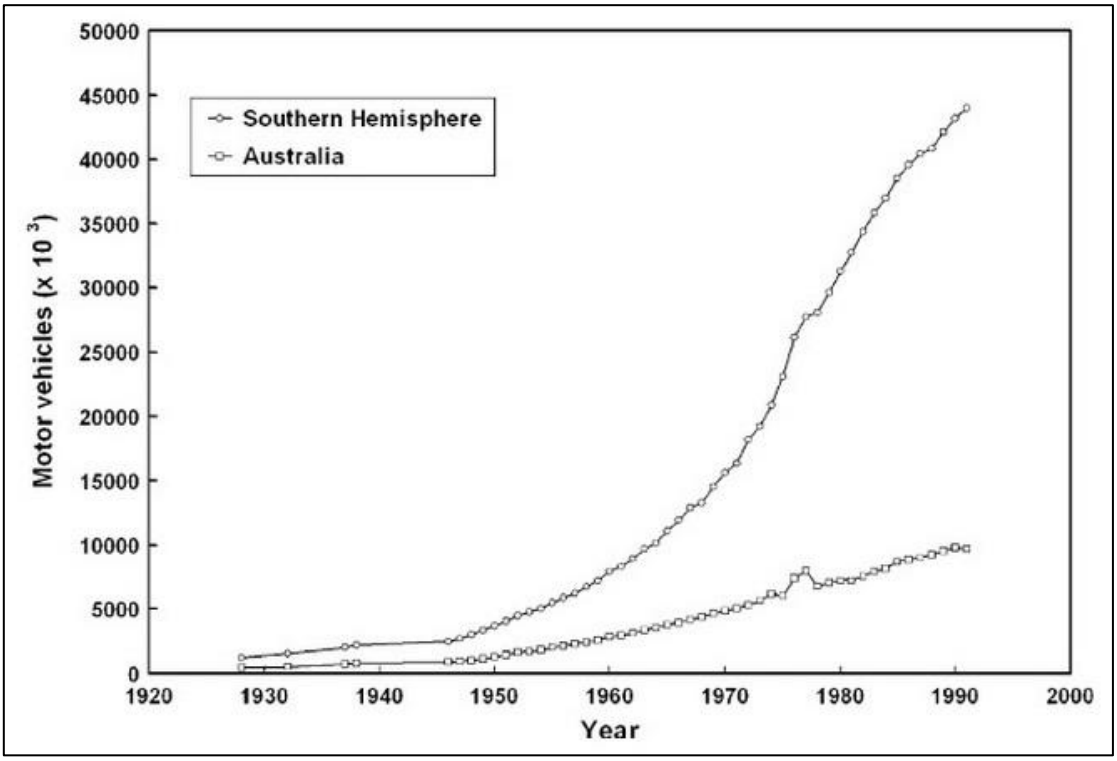

Figure 1.3 - Number of motor vehicles in Australia and the Southern Hemisphere, 1928–1991. Source of data: Statistical Office of the United Nations and its successors (1949–1955, 1957, 1959–1961, 1963–1979, 1981, 1983, 1985, 1988, 1992–1997, 1999–2002). The data were obtained by summing individual returns for all Southern Hemisphere countries. (Cook and Gale, 2005).

1.7 LEAD AS AN ADDITIVE IN PAINT IN AUSTRALIA

The Department of the Environment (2012) indicates that before 1970, paints containing high levels of Pb were used in many Australian houses. The recommended amount of Pb in domestic paint has declined from 50% before 1965, to 1% in 1965. In 1992, it was reduced to 0.25%, and in 1997 it was further reduced to 0.1%.

1.8 LEAD AS AN ADDITIVE IN PETROL AND PAINT IN THE UNITED STATES

Between 1910 and 1990, US paint and petrol (gasoline) additives accounted for a combined total of 10–12 million metric tonnes (MT) of industrial usage of Pb. Han et al. (2002) estimated that by the year 2000, the cumulative global industrial production of Pb has been about 235 million MT. Thus about 5% of the several thousand year global history of anthropogenic Pb production (Figure 1.4) was used in paint or petrol in the US during the 20th century.

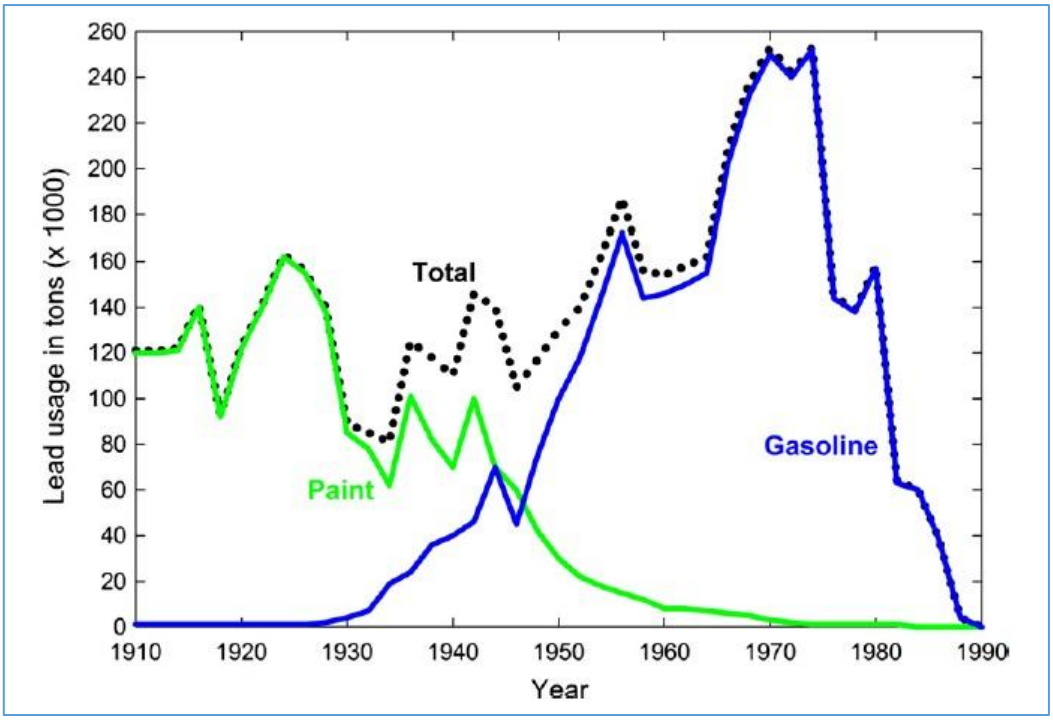

Figure 1.4 - History of Pb usage in paints and in gasoline during most of the 20th century, showing the early dominance of Pb-based paints followed by the boom in transportation which resulted in a high use of leaded gasoline (after Mielke et al., 1999). The decline after the mid-1970s was due controls put into place to eliminate leaded gasoline (Source = Laidlaw and Filippelli, 2008).

1.9 CONCEPTUAL MODEL OF THE DISPERSAL OF LEAD FROM VEHICLES TO URBAN SOIL

Pb in urban soils alongside roadways has accumulated with the highest concentrations adjacent to roadsides due to the former use of Pb in petrol (Figures 1.5 and 1.6). Soil Pb concentrations decay exponentially with distance from the roadway. Pb in soils within non-smelter urbanized areas is primarily derived from a mixture of Pb from paint and gasoline with a ratio that is spatially variable dependent on the proximity of roadways and/or age, condition, and maintenance of homes with exterior Pb paint (Wu et al., 2010).

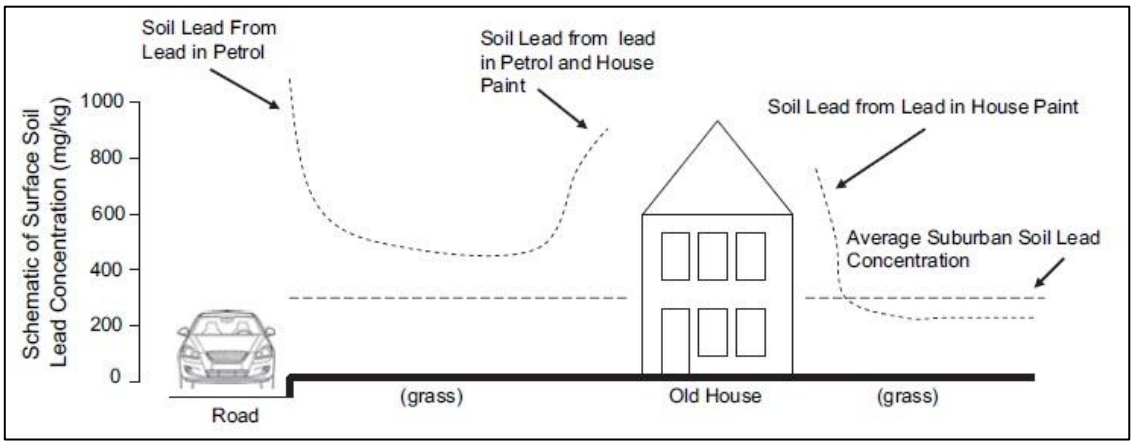

Figure 1.5 - A schematic cross-section through a residential suburban setting demonstrating typical urban soil Pb patterns (from Laidlaw and Taylor, 2011).

1.10 URBAN SOIL LEAD CONTAMINATION PATTERNS

1.10.1 Roadside Soil Lead Patterns

Figure 1.6, below, displays the typical urban and suburban patterns of average Pb concentrations with distance away from the roadway in the US. It shows that soil Pb concentrations decay exponentially with distance away from the roadway, with soil Pb concentrations significantly higher in UAs compared to suburban areas. This occurs because most of the Pb emitted from automotive exhausts was deposited close to the road with less

deposited as distance increases away from the roadside (Labelle et al., 1987). This general pattern has also been observed internationally (Farooq et al., 2012).

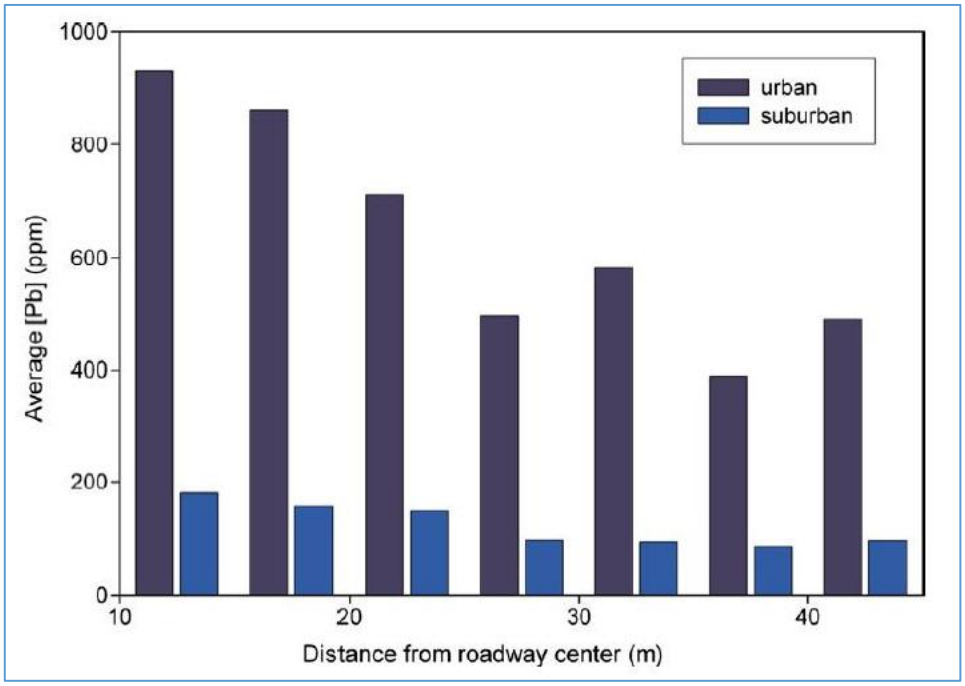

Figure 1.6 - Average Pb concentrations in surface soil as a function of distance from the roadway using the urban and suburban transects from Indianapolis, Indiana (Laidlaw and Filippelli, 2008). The decrease away from the roadway source is apparent. More important are the significantly higher values in the urban transect, even at distances up to 42.5 m from the road centre, beyond the range of direct deposition of Pb particulates from the combustion of leaded gasoline. Additionally, the significant near-roadway loading of surface soils in the urban transect is reflective of higher daily traffic volumes and much greater duration of the urban roadway as an important traffic artery.

1.10.2 Citywide Soil Lead Maps

Maps of soil Pb concentrations in major UAs typically display a 'bullseye' pattern with soil Pb concentrations highest in the city centres decreasing with distance from the city centre. Examples of this soil Pb pattern are depicted in the following soil Pb maps of London (Figure 1.7) and New Orleans (Figure 1.8). The British Geological Survey has also observed this pattern in other cities in Britain (British Geological Survey, 2012). This pattern of Pb distribution in urban city soils is very consistent globally (Laidlaw, 2014). Given the low mobility of Pb in soil, all of the Pb that accumulates on the surface layer of the soil is retained

within the top 20 cm (Laidlaw, 2001; Mielke et al. 1983). Studies of the atmospheric deposition from Pb mining in Port Pirie and Mount Isa (Taylor et al., 2010, 2013; Mackay et al., 2013) suggest that Pb (and other metals) accumulates in the soil surface (0-2 cm) layer, the portion of soil with which children are most likely to interact. The half-life of Pb in surface soils has been estimated to be approximately 700 years, thus, without corrective action, Pb dust will persist for many generations (Semlali et al. 2004).

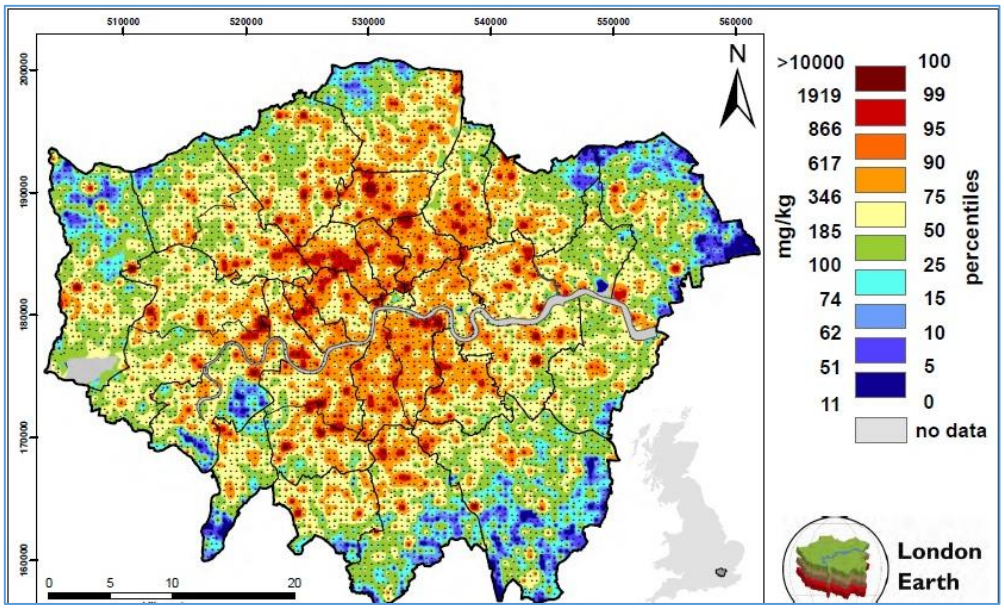

Figure 1.7 – Map of soil Pb concentration in London, England (UK) (Source: British Geological Survey, 2012).

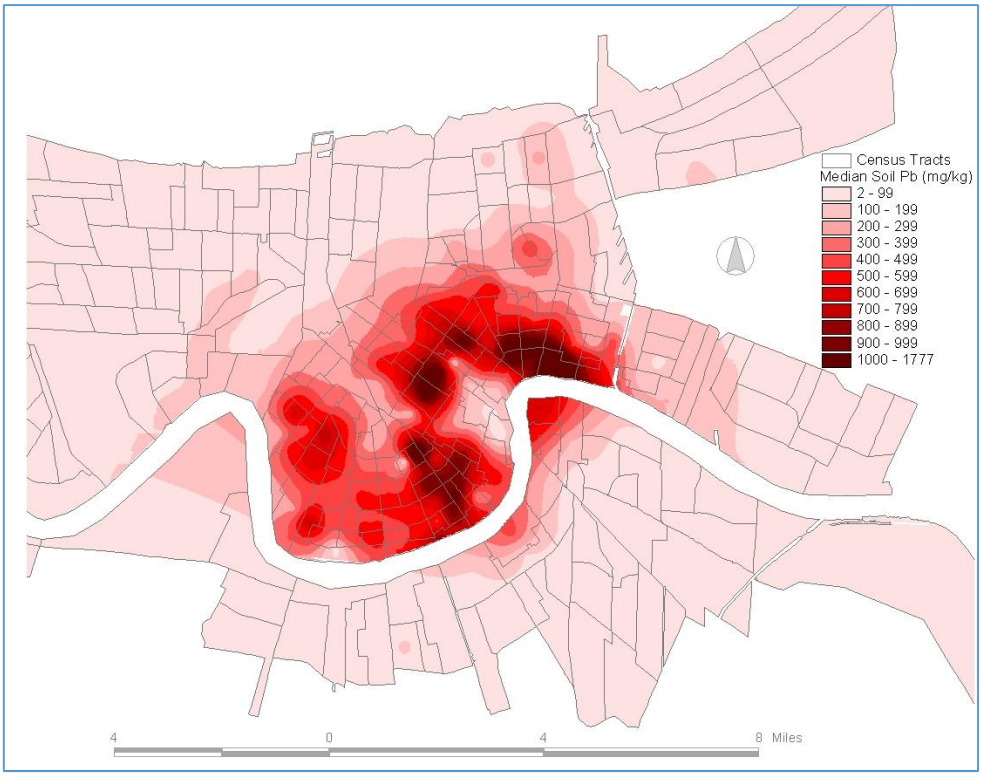

Figure 1.8 – Map of soil Pb concentration in New Orleans, Louisiana (Source: Mielke et al, 2001).

An enormous quantity of Pb was emitted into UAs when Pb was used as a fuel additive. For example, in New Orleans, vehicle traffic was responsible for an annual Pb emission of 50×10^9 μg of Pb dust per 0.1 mile (0.16 km) during the peak use of leaded petrol (gasoline) on an arterial street (Mielke et al., 2001). The higher volume of vehicle traffic in large UAs emitted higher quantities of Pb aerosols than the lower volume of traffic in smaller UAs. As a result, the deposition and storage of Pb in soils in larger cities is higher than the deposition and storage of Pb in soils of smaller towns (Mielke et al., 2010). Traffic and building-age related variables are similarly indicated as important variables for predicting soil Pb concentrations. Sutton et al. (1995) observed that homes built before 1920 were 10 times more likely to have soil Pb content ≥500 ppm compared to post-1950 homes. In 2002, after Pb additives to gasoline had been completely phased out, Lejano and Ericson (2005) analysed soil around Pacoima, California and found that both total and bio-available Pb were markedly

higher in areas close to major highways. Wu et al. (2010) collected 550 surface soil samples from south central Los Angeles and found that mean total and bio-available Pb concentrations were highly correlated (r=0.96); Pb concentrations near freeways and major arterials were significantly higher than soils collected at other locations. The implication of these studies is that there is an enormous reservoir of bio-available Pb in older urban soils.

1.11 SOIL LEAD RESERVOIR COMPARED TO INTERIOR LEAD DUST RESERVOIR

Soils contain an enormous reservoir of Pb compared to that of home interior dust. Mielke calculated the median soil Pb loadings (Pb mass/unit area) of small and large cities in New Orleans and Minnesota (Mielke, 1993). In large cities, soil Pb loading ranged from 7,700 $\mu g/m^2$ to 32,300 $\mu g/m^2$ (Mielke, 1993). The result of this elevated soil Pb loading is that soils have a high capacity to poison children via hand to mouth activity.

Mielke et al. (2007) devised a procedure, which he named PLOPS, for assessing the Pb loading ($\mu g/m^2$) on children's hands using soils with various Pb concentrations. Mielke et al. (2007) tested PLOPS on soils of various soil Pb concentrations. The procedure showed that as soil Pb concentrations increased, hand Pb loading increases (Figure 1.9). The PLOPS results provide insight into the potential of soil for transferring Pb directly via hand-to-mouth behaviour.

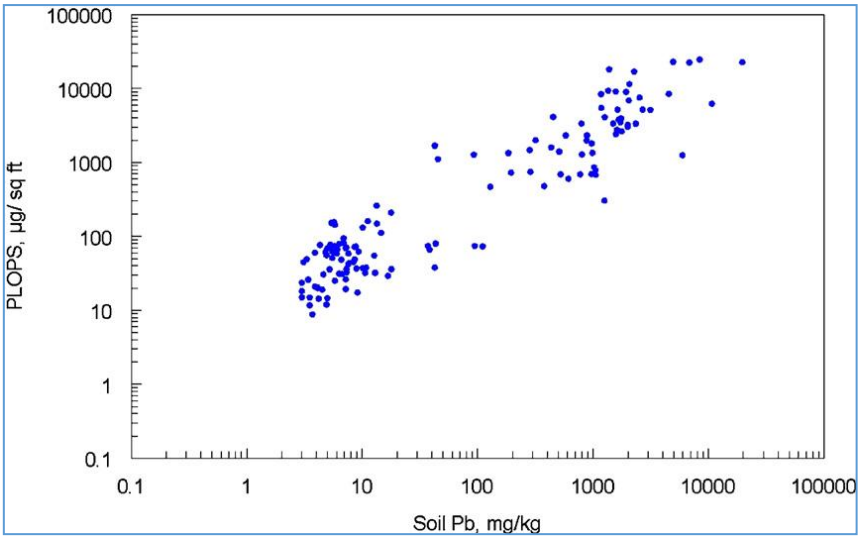

Figure 1.9 - Log–log scatter plot of soil Pb concentrations (mg/kg) vs. PLOPS loading value (µg/ft^2) (n=136). The values toward the upper right of the figure are for the initial soil Pb and associated PLOPS results. The values in lower left are for the new soil cover from the Bonnet Carre Spillway.

1.12 LEAD CONCENTRATIONS MAXIMA DURING SUMMER AND AUTUMN

Summer and autumn maxima of atmospheric Pb have been observed in Washington D.C. (Green and Morris, 2006), (Melaku et al. 2008), Boston (USEPA, 1995), New York (Billick et al., 1979), and Chicago (Paode et al., 1998). In Boston (USEPA, 1995), modelled Pb levels for air, floor dust and furniture dust. All the studies had an atmospheric Pb maxima in July. In Jersey City, New Jersey, Yiin et al. (2000) observed that windowsill Pb loadings (mass/unit area - µg/m^2) were most correlated with PbB concentration. The variation of dust Pb levels for floor Pb loading, windowsill Pb loading, and carpet Pb concentration were consistent with the variation of PbB levels, showing the highest levels in the hottest months of the year (June to August). In New Jersey (Edwards et al., 1998) found that the mean summertime household dust loadings of Pb were 68% higher than mean winter household dust loadings. Edwards et al. (1998) also observed that the dust mass deposition rate of Pb in summer (0.37 ± 0.13 µg/cm^2/day) was almost twice as great as in winter (0.22 ± 0.13 µg/cm^2/day). In Mexico City, Rosas et al. (1995) observed that during rainy seasons of the year, particulate matter less than

10 micron (PM_{10}) dust was settled and atmospheric Pb concentrations were lower; during seasons with low rainfall PM_{10} and atmospheric Pb concentrations increased. Interior summertime maxima Pb loadings were also observed inside the interior of a home in Northern England (Al-Radady et al., 1994). Al Radady et al. (1994) observed that between April and July (spring - summer), dust Pb loading rates increased on the walls (from 0.49 to 0.89 $\mu g/m^2$ per day), furniture (from 1.84 to 2.41 $\mu g/m^2$ per day), curtains (from 2.55 to 4.45 $\mu g/m^2$ per day), and window sills (from 2.57 to 5.86 $\mu g/m^2$ per day).

1.13 SUMMER AND AUTUMN CHILDREN'S BLOOD LEAD SEASONALITY

The summer and autumn maxima of atmospheric Pb presented in the previous section are consistent with summer and autumn child PbB seasonality maxima previously observed in Boston, Massachusets (USEPA, 1995), Chicago, Illinois (Blanksma et al., 1969), Connecticut (Stark et al., 1980), Indianapolis, Indiana (Laidlaw et al. 2005), Jersey City, New Jersey (Yiin et al., 2000), Lansing, Michigan (Hunter, 1978), Los Angeles, California (Rothenberg et al., 1996), Milwaukee, Wisconsin (USEPA, 1996), New Jersey (New Jersey Department of Health, 2007), New York State (Haley and Talbot, 2004), New York City (Billick et al., 1979) and Syracuse, New York (Johnson et al., 1996). The summer maxima in children's PbB was also observed in Birmingham, England (Betts et al., 1973). Havlena et al. (2009) observed that PbB levels followed a seasonal pattern with maxima in the summer and autumn. Havlena et al. (2009) observed that particulate matter less than 2.5 microns ($PM_{2.5}$) correlated with the seasonal variation in 10 month old children's PbB levels.

1.14 LEAD IN THE URBAN ATMOSPHERE – THE POST-LEADED PETROL (GASOLINE) ERA

The assumption that soil Pb is being re-suspended and is responsible for a large portion of the Pb in the atmosphere is supported by isotopic analysis of atmospheric Pb in Yerevan, Armenia (Kurkjian et al. 2003). This study indicated that following elimination of the use of Pb in gasoline, 75% of atmospheric Pb in the atmosphere was derived from re-suspended soil. Similarly, Kamenov (2008) analysed Pb isotopic ratios of teeth in Sofia Bulgaria and found the remarkable Pb isotopic composition similarity between the teeth Pb and the Pb additive used in gasoline in the local soils. The study concluded that soil and/or soil-born dust inhalation and/or ingestion are the most probable pathways for incorporation of soil Pb in the local population.

Harris and Davidson (2005) calculated that in the Southern California Air Basin (SOCAB), Pb particles deposited during the years of Pb additives being used in gasoline are being re-suspended into the atmosphere and responsible for generating approximately 54,000 kg of airborne Pb each year. This study used an average soil Pb concentration of 79 mg/kg as an input into their re-suspension model, while Wu et al. (2010) has calculated that the median soil Pb concentration in Los Angeles is 180 mg/kg, thus Harris and Davidson's (2005) SOCAB Pb re-suspension estimate may be conservative. Nevertheless, Harris and Davidson (2005) concluded that soil contamination contributes most of the total airborne Pb currently measured in the SOCAB and is likely to continue to do so for many years. Sabin et al. (2006) observed that approximately 12 MT of Pb (95% C.I. = 6–18) are being deposited from the atmosphere annually in the Los Angeles watershed.

Pb dust in exterior urban environments can result in elevated Pb loading of both interior and exterior contact surfaces (Caravanos et al., 2006 a,b). Pb loading (mass/unit area - $\mu g/m^2$) is well known to correlate with urban children's PbB levels. However, exterior Pb loading is not routinely measured in US cities, and is likely a better measure of risk to children PbB levels than air Pb concentrations (mass/volume - $\mu g/m^3$). Caravanos et al. (2006b) demonstrated that exterior Pb loading in the five boroughs of New York City was highly elevated when compared to the United States Department of Housing and Urban Development (USHUD)/United States Environmental Protection Agency (USEPA) indoor Pb in dust standard of 40 $\mu g/ft^2$ (3.7 $\mu g/m^2$). Caravanos et al. (2006b) measured the following median exterior dust loadings in New York: Brooklyn (730 $\mu g/ft^2$), Staten Island (452 $\mu g/ft^2$), the Bronx (382 $\mu g/ft^2$), Queens (198 $\mu g/ft^2$) and Manhattan (175 $\mu g/ft^2$). In a related study, Caravanos et al. (2006a) demonstrated how exterior particulate Pb can accumulate rapidly on interior surfaces. They observed that interior settled dust in a Pb-free room with a window slightly open exceeded the USHUD/USEPA indoor Pb in dust standard of 40 $\mu g/ft^2$ within a 6 week period.

1.15 LEAD SOURCE IDENTIFICATION USING LEAD ISOTOPES

Gulson et al. (1981) used Pb isotopes to conclude that the primary source of Pb in Adelaide Australia surface soils was from leaded petrol. Using Australia data from Donovan (1996), Gulson et al. (2013) measured Pb isotopic and Pb concentration measurements from children's blood, floor dust wipes, soil, drinking water and paint from 24 dwellings where children had previously recorded PbB levels ≥15 μg/dL in an attempt to determine the source(s) of their elevated PbB. Gulson et al.'s (2013) results indicated that there was a strong isotopic correlation of soils and house dust (r=0.53, 95% CI 0.20–0.75) indicative of a common source(s) for Pb in soil and house dust. Using Pb isotopes, Adgate et al. (1998a)

found that about half of the Pb in house dust of 10 homes in Jersey City, originated from sources outside the home, such as soil. Other studies have concluded that Pb sourced from Pb paint and gasoline can compose a significant fraction of Pb in soils. Clark et al. (2006) analysed Pb isotopes and concentrations in 103 soil samples (88% > 400 mg/kg) collected in surface soils of Roxbury and Dorchester, Massachusetts. Clark et al. (2006) concluded that there were two primary sources of Pb in the surface soil – Pb sourced from gasoline and paint, with Pb based paint contributing between 40% and 80% of the Pb. At homes near the Broken Hill mine site in Australia, a strong correlation (r = 0.95) was obtained between the Pb isotopic ratio of PbB samples and dust-fall accumulation, demonstrating the role that Pb bearing atmospheric particulates can have on PbB levels (Gulson et al., 1995).

Lead isotopes have been successfully used to examine the source of Pb in children's blood. In Torreon, Mexico, Pb isotopic ratios of the urban dust and soil, aerosols, and children's PbB were indistinguishable from each other (Soto-Jime´nez and Flegal, 2011). The source of Pb in children's blood has also been identified in a limited number of small Pb isotope studies (Rabinowitz, 1987; Gwiazda Smith, 2000), however its use has some inherent shortcomings (Rabinowitz, 1995; Duzgoren-Aydin and Weiss, 2008). Rabinowitz analysed Pb isotopes from blood of three patients with highly elevated PbB ranging from 66 µg/dl to 2,480 µg/dl. Lead isotopes were also analysed in faecal material of the children and in the air, water and soil in or near the children's homes. Pb paint appeared to be the main source of Pb in the children's blood (very high PbB levels). Rabinowitz (1987) concluded that childhood exposure from old residential Pb paint *and soil* appears to be the most intractable sources of Pb. He also concluded that in the absence of Pb paint, the Pb in urban soils and household dust have nearly identical isotopic compositions and are the product of decades of accumulated fallout.

1.16 SYNCHROTRON ANALYSIS - A NEW METHOD FOR ASSESSING SOURCES OF LEAD

Synchrotron analysis is a promising new technique that can be used to analyse the source of Pb in dust, soil and paint. Pingitore et al. (2009) used synchrotron-based XAFS (x-ray absorption fine structure) to quantify the Pb species in the air of El Paso and found that Pb-humate was the dominant form of Pb in contemporary El Paso air and was the major Pb species in El Paso soils. Consequently, Pingitore et al. (2009) concluded that in El Paso soil was the dominant source of Pb in the air, which was being re-suspended into the atmosphere (Laidlaw and Filippelli, 2008; Laidlaw et al., 2012).

Other recent synchrotron studies of Pb in house dust and soil were undertaken by Rasmussen et al. (2011), MacLean et al. (2011) and Walker et al. (2011). In Canada during the winter when soils are generally covered with snow, Rasmussen et al. (2011) collected 12 house dust samples from homes of varying dates of construction (1880 to 2000), which were analysed for Pb speciation (EXAFS (eight samples) and XANES (four samples)). The study concluded that the compounds were sourced from Pb paint and possibly soils. MacLean et al. (2011b) analysed four house dust samples using Synchrotron based XAFS, micro-X-ray fluorescence (μXRF), and micro-X-ray diffraction (μXRD) in four Canadian homes aged 9, 10, 28 and 105 years old. Source interpretation was complex due to various Pb compounds detected, however, soil, Pb paint and solder were suggested as possible sources. In another study, MacLean et al. (2011a) analysed a house dust sample and concluded that Pb paint and soil were the sources of Pb. Walker et al. (2011), performed synchrotron Pb speciation analysis (μ-XRF analysis and μ-XRD) in house dust samples collected in garden soil (Pb=652 mg/kg), from a living room (Pb=243 mg/kg) and bedrooms (Pb=5,094 and 14,032 mg/kg) in a Canadian home. The results indicated that there was a greater influence of exterior metal

sources (i.e. soil) in living room dust, and a greater influence of interior sources, specifically home renovation, on the metal signature of the dust collected in the bedrooms. Each of these studies concluded that Pb sourced from soil and paint were both present in the analysis of Canadian homes containing Pb paint.

1.17 SOIL LEAD EXPOSURE

There are two general routes of exposure to *soil Pb dust* in children – incidental ingestion and inhalation. Dermal absorption is not thought to be a significant route of exposure. Indoors, Pb dust settles on surfaces such as floors (and toys). Children then incidentally ingest Pb that adheres to their hands through hand to mouth activity (thumb sucking) (Ko et al., 2007). In outdoor environments children incidentally inhale and ingest Pb in soil dust, either directly from contact with the soil or by touching contact surfaces such as playground equipment where Pb has been deposited by re-suspension or deposition (Taylor et al., 2013). Stanek and Calabrese (1995) estimated soil ingestion rates for 64 children were 13 mg/day or less for 50% of the children and 138 mg/day or less for 95% of the children.

Kranz et al. (2004) estimated that Pb uptake via inhalation accounts for about 0.5-3% of an infant's PbB at 5 µg/dL. While inhalation appears to be a minor source of exposure in adults, Hodgkins et al. (1991) suggested that Pb absorption in the lung is more efficient than gastrointestinal absorption with a 10 to 1 ratio in absorption efficiency. Only soil dust particles between 0.5 and 10 µm are deposited in the alveoli, with the average soil dust particle size being about 2 µm in diameter (WHO, 2013).

Juhasz et al. (2010) observed and noted that the research literature indicates that as grain size decreases from a bulk soil, Pb concentration and bio-availability increases significantly. This suggests that bio-availability increases as grain size decreases possibly due to an increase in

surface area of the particles. This indicates that the Pb associated with re-suspended soils that is inhaled and ingested by children can have high concentrations of Pb which are more toxic and bio-available than that held within the bulk soil.

Semple et al. (2004) defines a bioavailable compound as *"...that which is freely available to cross an organisms cellular membrane from the medium the organism inhabits at a given time*." A bioaccessible compound is defined as "*...that which is available to cross an organisms cellular membrane from the environment, if the organism has access to the chemical. However, the chemical may be either physically removed from the organism or only bioavailable after a period of time....physically removed may refer to a chemical that is occluded in soil organic matter and hence is not available at a given time or that occupies a different spatial range of the environment than the organism*."

Re-suspension of soil typically occurs in the summer and autumn when soils are dry and evapotranspiration is at its maximum (Laidlaw et al., 2005; Laidlaw and Filippelli, 2008; Laidlaw et al., 2012). During such conditions, the finer fraction of soil becomes re-suspended when subjected to traffic turbulence or wind (Laidlaw et al., 2012). Given that Pb is typically enriched in this fine particle fraction (Bergstrom et al., 2011), this results in a soil dust Pb concentration that is up to 5 times higher than the bulk soil from which it originated (Juhasz et al., 2011). Re-suspended soil dust enters the atmosphere and then penetrates home interiors where it can settle on to contact surfaces (Kranz et al., 2004; Layton and Beamer, 2009) such as floors and toys etc, where after it is available for accidental exposure.

In addition to soil re-suspension, penetration, and settling of soil dust onto contact surfaces (floors and toys etc.), soil is also tracked-in to the interiors of homes via human feet and pets

feet and fur. Hunt and Johnson (2012) quantified the soil track-in process using laser beams and showed that step-on impacts produced a temporary increase in particle levels at various lateral distances and heights from the contact point. With increasing distance and height from the step-on contact point, the level of suspended particles after successive step-on events decreased markedly. Hunt et al. (2006) investigated rates of dry and wet soil deposition on indoor hard surface flooring as a result of mass transfer from soiled footwear. They observed that under repeated tracking conditions, with multiple soil incursions, widespread floor surface contamination was possible.

A conceptual diagram depicting the movement of contaminated soil and airborne particulates into a residence is presented in Figure 1.10. This conceptual diagram shows that the imported soils mix with organic matter in floor dust and become redistributed indoors occurs via re-suspension, with some losses due to cleaning and ventilation by building air circulation systems.

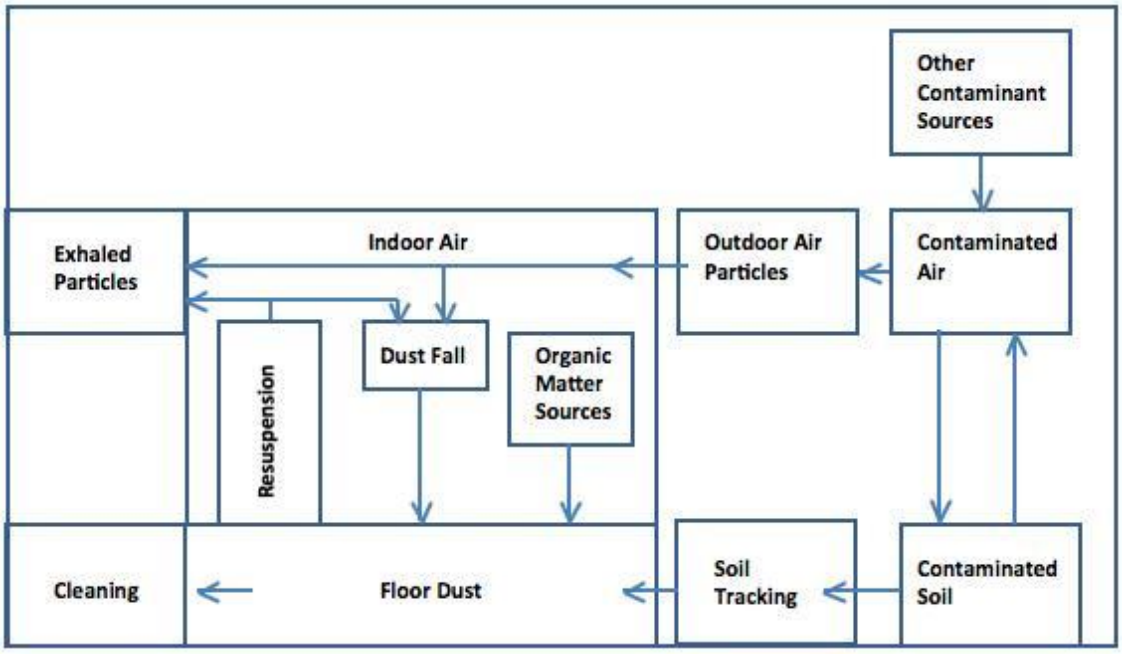

Figure 1.10 - Conceptual diagram depicting the movement of contaminated soil and airborne particulates into a residence, subsequent mixing with organic matter in floor dust,

redistribution indoors via re-suspension, and removal by cleaning and exhalation with building vented air (figure adapted from Layton and Beamer, 2009).

1.18 SOIL PB IN THE SYDNEY STUDY AREA

The most recent and most complete survey of soil Pb concentrations in the Sydney Basin was conducted by Birch et al. (2011). A map depicting the Post Extraction Normalizing Procedure (PEN) normalized soil Pb concentration is presented below in Figure 1.11. This procedure normalizes the soil Pb concentration to the 63 µm grain size fraction. This fraction is important because smaller grain sizes adhere to children's hands (Juhasz et al., 2011). This map shows that the soil is most contaminated immediately north, east and south of Sydney, and decreases with distance from the city centre. Comprehensive soil Pb maps have not been completed in any of the other major Australian cities. However, there have been some ad-hoc studies which typically show that soils in urban inner city areas are contaminated with Pb (Olszowy et al., 1995; Laidlaw and Taylor, 2011).

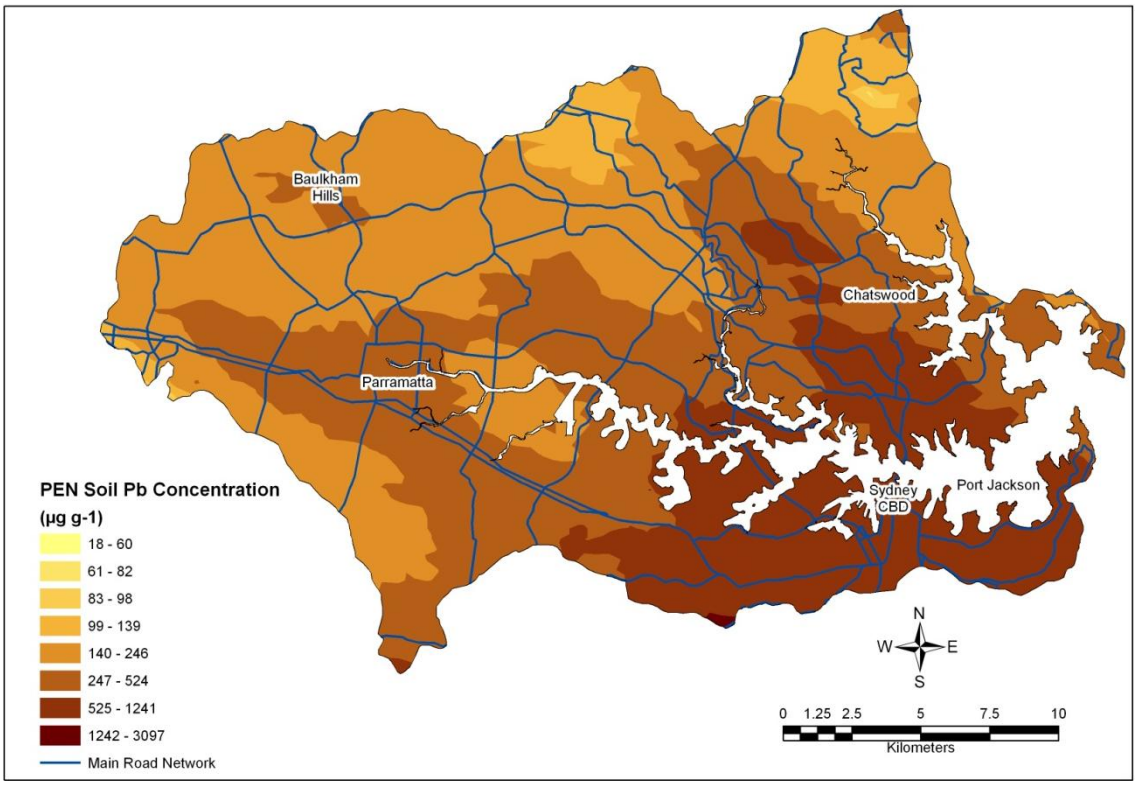

Figure 1.11 – PEN Normalised Soil Pb concentration in the Sydney Basin, Australia (Birch et al., 2011).

1.19 AIMS

The primary aims of this thesis are addressed via eight peer-reviewed research papers.

Specifically this thesis addresses the following hypotheses: (1) that Pb in soil dust plays a major role in elevating children's PbB levels, particularly at low, chronic exposure levels; (2) petrol-derived Pb can be a major source of Pb in urban soils, house dust and children's PbB levels; (3) Re-suspended urban Pb contaminated soil dust drives urban atmospheric Pb concentrations and seasonal variations in children's PbB levels; (4) Urban soils in Australian and America's old inner-city areas are contaminated with Pb, which poses a potential hazard to children's health.

1.20 THESIS OUTLINE

The primary aims of this thesis are detailed via eight peer-reviewed research papers (Chapters 2 to 4), which can be divided broadly into soil Pb and children's exposure in the Australian context (Chapter 2), historical petrol Pb emissions and soil Pb studies in California and 90 US urban areas (Chapter 3), and spatial and temporal associations between soil Pb and PbB in urban children (Chapter 4). The following sections provide a brief background and précis for each thesis chapter.

Chapter 2 contains two papers that evaluate soil Pb and PbB exposure in the Australian context, and one letter, which is a response to Gulson's (2014) comments on the Laidlaw et al. (2014a) paper. The Laidlaw and Taylor (2011) paper reviews the literature for evidence that Pb in soil in urban areas of Australia is poisoning urban children. The Laidlaw et al. (2014a) paper evaluates temporal and spatial soil Pb exposure dynamics in 5 western Sydney homes for 15 months between November 2010 and January 2012.

Chapter 3 estimates the mass of petrol-derived Pb emitted historically into the atmosphere in 90 US urban areas and major California cities (Mielke et al., 2010; Mielke et al., 2011). Historical soil Pb studies in the US and California are also presented and discussed (Mielke et al., 2010; Mielke et al., 2011).

Chapter 4 demonstrates that soil Pb is spatially and temporally associated with children's PbB levels. In the first paper, Laidlaw et al., (2012) documented the associations between atmospheric soil Pb and children's PbB levels in Birmingham, Alabama Chicago, Illinois Detroit, Michigan and Pittsburgh, Pennsylvania. It also evaluated weekly variations in atmospheric soil and Pb in the four cities. The second paper, Zahran et al., (2013a) evaluates the association between atmospheric soil Pb, atmospheric Pb and children's PbB in Detroit, Michigan. The third paper, Zahran et al., (2013b), documented the spatial association between soil Pb and children's PbB levels in New Orlean's, Louisiana and assesses optimum sample locations for assessing children's health risk due to exposure to Pb in soils. The fourth paper, Filippelli and Laidlaw (2010) argue that our recent research regarding the causes of swings in seasonal PbB levels present a new paradigm of the exposure pathway of children to Pb and point to a relatively simple and cost-effective way toward reducing the Pb load for urban youth. Chapter 5 contains the overall thesis discussion and integrates the findings of the research, with the final conclusions being presented in Chapter 6.

1.21 STUDY REGION

Studies were conducted in two countries – Australia and the United States.

The study region consists of the following countries and locations:

- Australia:
    - Australian metropolitan cities (review);

    - Inner west of Sydney, NSW;
- United States;
    - California ;
    - Birmingham, Alabama ;
    - Chicago, Illinois ;
    - Pittsburgh, Pennsylvania ;
    - Detroit, Michigan ; and
    - New Orleans, Louisiana.

The field research was conducted in the inner west of Sydney because Laidlaw and Taylor's (2011) review paper indicated that the soils and vacuum bag dust Pb concentrations in the inner west of Sydney were highly elevated. Research conducted in these locations would therefore allow testing of multiple hypotheses about Pb dust dynamics. I had contacts in the area that allowed me to conduct the study in residential homes, which was pivotal to one of the thesis aims (see below).

Historical Pb emission estimates for US cities were created to understand historical exposures and to estimate the amount of Pb emitted into urban soils. The United States individual city Pb emissions estimates used in the Mielke et al. (2010) and Mielke et al. (2011) were calculated using data only available in the United States. Similarly, the USEPA IMPROVE atmospheric data used in the Laidlaw et al. (2012) and USEPA IMPROVE atmospheric data and the Detroit Child PbB Database used in the Zahran et al. (2013a) study were also only available in the United States.

CHAPTER 2

Petrol derived Pb in urban surface soils is a major source of Pb in house dust and has the potential to poison children in the inner cities of Australia.

Chapter 2 contains three papers that evaluate soil Pb and PbB exposure in the Australian context.

The Laidlaw and Taylor (2011) *Environmental Pollution* paper was undertaken at the beginning of my candidature to help focus my field research efforts. This work and previous research indicated that there remained ambiguity in the understanding of the predominant source of Pb inside urban homes. The Laidlaw et al., (2014a) *Environmental Pollution* paper attempted to better understand the source of Pb in urban homes by conducting a 15 month field research study of interior and exterior Pb dust dynamics and source identification. The Laidlaw et al. (2014b) paper contains Laidlaw et al.'s response to comments made by Brian Gulson (Gulson, 2014) about the Laidlaw et al. (2014a) paper.

Paper 1: "*Potential for childhood Pb poisoning in the inner cities of Australia due to exposure to Pb in soil dust."*

MAS Laidlaw, MP Taylor

Published in *Environmental Pollution* (2011).

The Laidlaw and Taylor (2011) *Environmental Pollution* paper summarised the literature about soil Pb and children's PbB levels in Australia. It concluded that soil Pb is highly contaminated in many urban areas and could potentially be a major source of exposure. It suggested that given known large sample dose-response relationships between soil Pb and PbB (Bickel, 2010;Mielke et al., 2007; Zahran et al., 2011), and given the soil Pb concentrations observed in cities such as Sydney (Birch et al, 2011, Laidlaw and Taylor,

2011), there remains a need for PbB monitoring because there is significant unknown health risk to urban children exposed to Pb.

Author Contributions (Laidlaw and Taylor, 2011):

MAS Laidlaw: 85%

Concept, literature review, writing of text.

MP Taylor: 15%

Editing of text.

Paper 2: "*Source identification of temporal Pb sources in domestic homes."*

Published in *Environmental Pollution.*

MAS Laidlaw, S Zahran, N Pingitore, J Clague, G Devlin, MP Taylor

The Laidlaw et al. (2014a) *Environmental Pollution* paper evaluated temporal and spatial soil Pb exposure dynamics in 5 western Sydney homes for 15 months between November 2010 and January 2012. The principal aim of the paper was to determine the predominant source(s) of Pb inside typical western Sydney brick homes. Using Pb isotopes and X-Ray Absorption Spectroscopy (XAS), it was concluded that petrol derived soil Pb was the dominant source of Pb in the house dust (Laidlaw et al., 2013). The paper also concluded that Pb was migrating from exterior soil to the interior of the homes.

Author Contributions (Laidlaw et al., 2014a):

MAS Laidlaw: 60%

Concept, sampling, study design, writing, statistical analysis and editing.

S Zahran: 15%

Statistical analysis, charts, writing, editing**.**

N Pingitore: 2%

Synchrotron interpretation

J Clague: 2%

Synchrotron interpretation, data extraction

G Devlin: 1%

Synchrotron operation.

MP Taylor: 20%

Mentoring, study design, editing of text.

Paper 3: "*Response to Brian Gulson's - Comments on: Identification of lead sources in residential environments: Sydney Australia."*

The Laidlaw et al. (2014b) paper contains Laidlaw et al.'s response to comments made by Brian Gulson (Gulson, 2014) about the Laidlaw et al. (2014a) paper.

MAS Laidlaw, S Zahran, N Pingitore, J Clague, G Devlin, MP Taylor

Author Contributions (Laidlaw et al., 2014b):

MAS Laidlaw: 60%

Concepts, writing, editing

S Zahran: 5%

Editing

N Pingitore: 5%

Editing

J Clague: 0%

G Devlin: 0%

MP Taylor: 30%

Concepts, writing, editing

Chapter 2 - Paper 1: "*Potential for childhood Pb poisoning in the inner cities of Australia due to exposure to Pb in soil dust.*"

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Review

Potential for childhood lead poisoning in the inner cities of Australia due to exposure to lead in soil dust

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Previous use of Pb in gasoline and Pb in exterior paints in Australia has contaminated urban soils in the older inner suburbs of large cities and the risks remain unconstrained.

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ABSTRACT

This article presents evidence demonstrating that the historical use of leaded gasoline and lead (Pb) in exterior paints in Australia has contaminated urban soils in the older inner suburbs of large cities such as Sydney and Melbourne. While significant attention has been focused on Pb poisoning in mining and smelting towns in Australia, relatively little research has focused on exposure to Pb originating from inner-city soil dust and its potential for childhood Pb exposures. Due to a lack of systematic blood lead (PbB) screening and geochemical soil Pb mapping in the inner cities of Australia, the risks from environmental Pb exposure remain unconstrained within urban population centres.

1. Introduction

In the United States (US) and Australia there is a substantial body of evidence of widespread soil lead (Pb) contamination in large inner-city areas (Mielke et al., 2007, 2010b; Olszowy et al., 1995) and in centres for metal mining and smelting (see Taylor et al., 2010). In this paper, US and Australian research related to urban soil Pb distributions, studies of the association between soil Pb and PbB, PbB screening practices and PbB prevalence are presented. The US soil Pb exposure research is presented alongside relevant Australian research because it provides direct parallels to the Australian situation. In addition, international urban soil Pb exposure prevention methods and the current urban Pb poisoning exposure paradigm are presented. Finally, this article presents a review of the evidence of toxicity from low-level exposure (<10 μg/dL) in children typically due to chronic soil Pb dust exposure and makes recommendations to improve the long-term public health of inner-city children in Australia.

2. Urban soil lead distribution – United States

In the US, motor vehicles used gasoline containing tetramethyl and tetraethyl Pb additives from the 1920s to 1995 (Mielke et al., 2010a). By the 1950s, Pb additives were contained in virtually all grades of gasoline. By 1986, when leaded gasoline underwent a rapid phase-down, 5–6 million metric tons of Pb had been used as a gasoline additive, and about 75% of this Pb was released into the atmosphere (Chaney and Mielke, 1986; Mielke and Reagan, 1998). Thus, an estimated 4–5 million tons of Pb has been deposited into the US environment by way of gasoline-fueled motor vehicles (Mielke, 1994). Accumulation of soil Pb created by leaded gasoline is considered proportional to highway traffic flow (LaBelle et al., 1987; Mielke et al., 1997). About the same quantity of Pb (5–6 million metric tonnes) was also used in production of Pb based paint in the US (Mielke and Reagan, 1998), which has added to the burden of environmental Pb in urban soils.

In the 1970s, the presumed dominant source of soil Pb contamination was Pb-based house paint (Ter Haar and Aronow, 1974). A subsequent study of garden soils conducted in metropolitan Baltimore, Maryland, began to raise questions about that assumption (Mielke et al., 1983). Soil around Baltimore's inner city buildings, predominantly unpainted brick, exhibited the highest

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amounts of Pb, while soils outside of the inner city, where buildings were commonly constructed with Pb-based paint on wood siding, contained comparatively low amounts of Pb. These findings indicated that Pb based house paint could not account for the observed pattern of soil Pb (Mielke et al., 1983).

The quantity and distribution of soil Pb have been studied in numerous places in the US (e.g. Mielke et al., 2010b; Laidlaw and Filippelli, 2008; Burgoon et al., 1995; USEPA, 1996, 1998). The U.S. cities exhibited the same distance decay pattern with high soil Pb concentration values in the inner city and decreasing contamination towards the outer parts of the urban area (Mielke et al., 1983; Laidlaw, 2010). Urban soil Pb patterns are well understood. As Fig. 1 indicates, soil Pb decays exponentially away from the roadside, with the concentration proportional to historical traffic volume (LaBelle et al., 1987; Filippelli et al., 2005; Lejano and Ericsson, 2005). A large percentage of the Pb emitted from automobiles has been deposited within approximately 50 m of the roadside. Soil Pb also decays exponentially with distance away from the house-side towards the roadside (see Fig. 1). In homes that used exterior Pb-based paint, the Pb in the house garden soil is a mixture of vehicular Pb and paint Pb (Linton et al., 1980), with automotive Pb concentrated in the finer grain size (<44 μm), which is susceptible to resuspension (Clark et al., 2006). Houses are impacted by vehicular Pb when exhaust emission particles came into contact with the house-side and are deposited in the soils adjacent to the house (see Fig. 1). This is evidenced by elevated Pb concentrations being found adjacent to brick homes facing roadways (Mielke et al., 1983; Olszowy et al., 1995).

3. Australian inner-city soil lead contamination

The problem of environmental lead exposure in children from paint and dust was first identified over a century ago by the Queensland doctors Gibson (1904) and Turner (1909). However, it appears that their warnings about environmental lead exposure went largely unheeded and leaded petrol was introduced in Australia in August 1932 (Cook and Gale, 2005). Unleaded petrol was introduced in 1996, and the Pb content of leaded petrol declined from 0.84 g/L in 1990 to 0.2 g/L in 1996, until it was banned in 2001 (Queensland Health, 2008). Lead levels in Australian paint were up to 50 % before the 1950s but thereafter several reductions were mandated bringing the allowable concentration to 0.1% in 1997 (Taylor et al., 2010).

A review of the literature indicates that some of the soils from the inner-city suburbs of Sydney, Australia, have become contaminated with a range of metals including Pb. It is likely that these soils have been contaminated due to the use of Pb in gasoline and Pb in exterior paints. The median and mean background soil lead concentrations in the Sydney region are 15.5 mg/kg and 21.3 mg/kg, respectively (Olszowy et al., 1995). In Balmain, an Inner Sydney suburb located 2.5 km north-west of Sydney, a soil Pb survey of 41 samples found that 68 % of residential housing samples exceeded

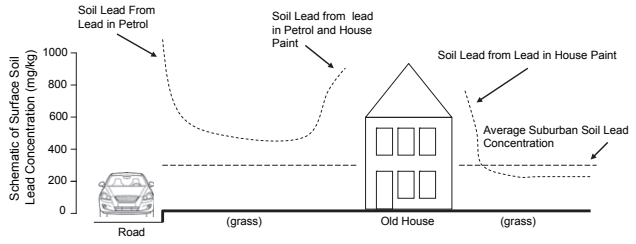

Fig. 1. A schematic cross-section through a residential suburban setting demonstrating typical urban soil Pb patterns (modified from Olszowy et al., 1995).

the National Environmental Protection Council (NEPC, 1999) 300 mg/kg residential soil lead guideline (Royal Prince Alfred Hospital and Central and Southern Sydney Area Health Service, 1988). Fett et al. (1992) analysed soil Pb concentrations at 18 homes in the inner Sydney suburbs and observed a median and mean "sink" (garden) soil Pb concentrations of 1237 mg/kg and 1944 mg/kg (range = 123 to 5407 mg/kg), respectively. In addition, at 24 inner Sydney suburban homes, Fett et al. (1992) observed a median and mean "play" area soil Pb concentration of 380 mg/kg and 627 mg/kg, respectively. Fifty-four % of all soil samples exceeded the NEPC 300 mg/kg residential soil Pb guideline (NEPC, 1999).

Skinner et al. (1993) collected seven soil samples from Bradfield Park (beneath the Harbour Bridge) and three samples at distances up to 350 m from the park. Four sites were sampled farther north at distances of 50–300 m from the major arterial Warringah Freeway in North Sydney. The median values for the two areas were 708 mg/kg (range = 19 to 1451 mg/kg) and 637 mg/kg (range = 216 to 1269 mg/kg), respectively. Olszowy et al. (1995) analysed 80 surface soil samples from residential properties in Sydney and found that about 40% of soil samples exceeded the residential 300 mg/kg soil Pb guideline. Snowdon and Birch (2004) analysed Pb concentrations in 274 soil samples in Iron Cove Catchment in Sydney (located approximately 2.5 km west of Sydney). They observed that 33% of the samples exceeded the NEPC (1999) guideline for Pb. Further, the mean bioavailability of the Iron Cove Catchment soils was 70 % using an EDTA extraction (Snowdon and Birch, 2004). Markus and McBrantney (1996) analysed 219 surface soil samples for Pb and other heavy metal concentrations in Glebe, a suburb located approximately 1 km south-west of Sydney, and found that greater than 50 % of Pb concentrations exceeded the 300 mg/kg residential soil Pb guideline. Cattle et al. (2002) reported that 41% of 807 surface soil samples in Glebe and Camperdown (located immediately west of Glebe) exceeded the 300 mg/kg residential soil Pb guideline. Cattle et al. (2002) also tested four geostatistical techniques to determine which methods were best able to delineate between soil Pb concentrations above and below the 300 mg/kg guideline in Camperdown and Glebe. Markus and McBrantney (2001) compiled a brief review of Australian soil Pb studies prior to 2001. Pb in Australian urban inner city soils is highly bioavailable (Snowdon and Birch, 2004) with bioavailability likely to be positively correlated with total Pb concentration (Wu et al., 2010). upon the NEPC (1999) 300 mg/kg residential soil guideline and a range of other studies showing a relationship between soil Pb and PbB. This suggests that soils in large areas of inner Sydney may potentially pose a toxic threat to children and adults (Table 1).

In addition to exterior soil Pb contamination in Sydney, dusts collected from vacuum bags, the interiors of some homes and ceiling cavities are also contaminated by Pb. For example, Gulson et al. (1995) collected vacuum bag bulk dust samples in five homes in Sydney (locations not indicated) which were analysed for Pb concentration. Results indicated Pb concentrations ranged between 460 and 2950 mg/kg, with a median of 1202.5 mg/kg and an average of 1344 mg/kg. Chattopadhyay et al. (2003) completed Pb dust sampling in 82 residential homes in the Sydney metropolitan area. Results showed statistically significant differences of Pb levels by region in Sydney but not for other metals. Large variations in Pb levels were found in household dust (Range = 16–16,600 mg/kg; Mean = 389 mg/kg; Median = 76 mg/kg), with the inner-west area associated with significantly higher Pb levels ($P < 0.001$) compared with other regions (Table 2). Chattopadhyay et al. (2003) also observed that household dust Pb levels have remained constant over the past decade despite substantial improvements in air quality. Gulson et al. (1995) collected ceiling dusts at 38 locations in the greater Sydney area. Results indicated median ceiling dust Pb concentrations of

Table 1
List of Sydney and Sydney Suburbs Soil Pb Studies.

Author(s)	Suburb	Outcome of soil lead assessment
Olszowy et al. (1995)	Sydney Region	Soil Pb Background: median = 15.5 mg/kg, mean = 21.3 mg/kg
Royal Prince Alfred Hospital and Central and Southern Sydney Area Health Service (1988)	Balmain (2 km north-west of Sydney)	68 % of 41 samples exceeded the 300 mg/kg guideline
Fett et al. (1992)	Sydney Suburbs	Pb median in garden soil = 1,237 mg/kg. 54 % of soil samples (all types) exceeded the 300 mg/kg residential soil Pb guideline. (n = 22 homes)
Skinner et al. (1993)	North Sydney (3 km north of Sydney)	Median value of two groups of samples were 708 mg/kg ($n = 10$; range = 19 to 1451 mg/kg) and 637 mg/kg ($n = 4$; range = 216 to 1269 mg/kg)
Olszowy et al. (1995)	Sydney Suburbs	40 % of 80 samples exceed 300 mg/kg guideline
Gulson et al. (1995)	Inner Sydney Suburbs	Five houses were sampled with 11 total samples collected (average = 1217 mg/kg, median = 1135 mg/kg and range = 37–3130 mg/kg).
Markus and McBrantney (1996)	Glebe (1 km southwest of Sydney)	50 % of 219 samples exceed 300 mg/kg residential soil Pb guideline
Snowdon and Birch (2004)	Iron Cove Catchment (2.5 km west of Sydney)	33 % of 274 samples exceed 300 mg/kg guideline
Gulson et al. (2006)	Sydney Suburbs (located at varying distances from major traffic thoroughfares in Sydney)	In a large study in Sydney, Gulson et al. (2006) observed that the Pb concentration in soil was a significant predictor for Pb in the house dustfall, and dustfall was a significant predictor of PbB concentrations.

1960 mg/kg in the industrial area ($n = 10$), 1022 mg/kg in the semi-industrial area ($n = 17$), 621 mg/kg in the non-industrial area ($n = 10$) and 145 mg/kg in the rural area ($n = 1$).

In Brisbane, Olszowy et al. (1995) observed that about 40% of soil samples from residential properties in old areas near busy roads exceeded the 300 mg/kg residential soil Pb guideline. In Adelaide, Tiller et al. (1987) analysed about 600 surface samples for Pb from a 90 × 20 km study area extending from the metropolitan area of Adelaide, South Australia, to rural areas. The Pb content of surface soils showed petrol-Pb emitted within Adelaide from automotive exhausts has measurably contaminated the rural landscape to about 50 km downwind of the city. Soil Pb concentrations in Adelaide (Tiller et al., 1987) were digested using EDTA and are not comparable to soil Pb concentrations in other Australian cities which are based on total extractable Pb concentrations. Gulson et al. (1981) measured lead isotopes in surface soil near Adelaide and concluded that Pb from gasoline was the main source of Pb in surface soil.

In New South Wales, Queensland, Victoria and South Australian cities, Olszowy et al. (1995) observed the following relative soil Pb gradient concentrations for various city areas: Old High Traffic > Old Low Traffic > New High Traffic > New Low Traffic > Rural. These findings are, in effect, similar to those of Mielke et al.'s (1997) work in the USA. Even in the old areas with low traffic flow in Brisbane, Sydney and Melbourne approximately 20% of samples were found to exceed the investigation threshold for Pb (Olszowy et al., 2005).

Soils in some areas of regional cities such as Newcastle are also contaminated with Pb. Devey and Jingda (1995) analysed Pb in 108 surface soil samples from public parks and playgrounds in Newcastle, New South Wales (NSW). This study found that soil Pb concentrations ranged from 25 to 2400 mg/kg and that 21% of samples had concentrations higher than the 300 mg/kg residential soil Pb guideline. This assessment excluded the areas of Boolaroo and Argenton, which have been severely impacted by the former Pasminco Pb smelter (now closed) (Willmore et al., 2006; NSW Environmental Protection Authority, 2003).

Table 2
Geometric mean interior house dust Pb concentrations in Sydney by region (after Chattopadhyay et al., 2003). Note that the Sydney mean Pb dust concentration is 389 mg/kg and the median is 76 mg/kg ($n = 82$).

Location	Geometric mean interior house dust Pb Concentration (mg/kg)
CBD and Eastern Suburbs	106
North Shore	66
Inner west	260
South West	110
North West	46
South	92

4. Emerging soil Pb exposure paradigm

The emerging PbB poisoning paradigm is that children in cities unaffected by Pb mining and smelting are also exposed to soil Pb dust, which can be traced to the use of Pb in gasoline and exterior Pb paint (Filippelli and Laidlaw, 2010). Contaminated dust is tracked into homes by shoes (Hunt et al., 2006), family pets, and also via resuspension and deposition of Pb dust, which penetrates interiors of homes and settles onto contact surfaces (Layton and Beamer, 2009; Laidlaw and Filippelli, 2008). Analysis of interior house dusts indicates that a large percentage of interior house dust most probably originates from outdoor soils (see Table 3). This illustrates the significance of the soil reservoir as significant potential exposure pathway for childhood Pb poisoning.

Fig. 2 shows a conceptual diagram depicting the movement of contaminated soil and airborne particulates into a residence, subsequent mixing by the organic matter in floor dust, redistribution indoors via resuspension, and removal by cleaning and exhalation with building vented air (Layton and Beamer, 2009). Once Pb has been tracked into homes, exposure to interior house dust then

Table 3
Estimates of the relative contribution of exterior soil to house dust (Paustenbach et al., 1997).

Environmental soil and dust Pb study	% House dust from soil
Hawley (1985)	>80
Thornton et al. (1985)	20
Camann and Harding (1989)	50
Fergusson and Kim (1991)	30–50
Calabrese and Stanek (1992)	20–78

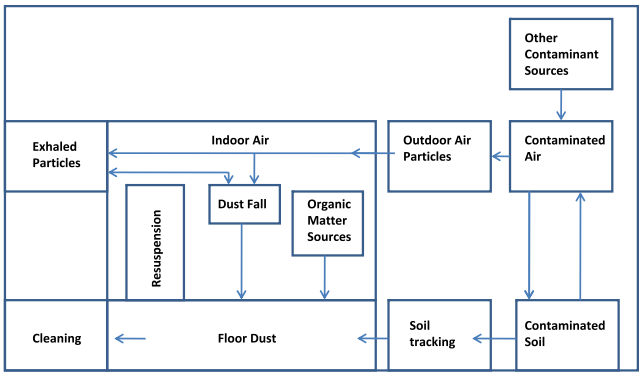

Fig. 2. Conceptual diagram depicting the movement of contaminated soil and airborne particulates into a residence, subsequent mixing with organic matter in floor dust, redistribution indoors via resuspension, and removal by cleaning and exhalation with building vented air (Source – adapted from Layton and Beamer, 2009).

occurs via hand-to-mouth behaviour. Acute exposure occurs typically via ingestion of paint chips from indoor and outdoor Pb paint, which is most prevalent when children are aged approximately 18–24 months. At this age children are at an exploratory phase of their development and the ingestion of non-nutrient substances may result in accidental ingestion of poisons (e.g. Pb rich soil, paint chips, or paint from toys), leading to clinical or sub-clinical toxicities (Ko et al., 2007). In addition, another potentially important exposure pathway for Pb into humans may be via ingestion of contaminated vegetables (Finster et al., 2004; Kachenko and Singh, 2004, 2006).

5. Association between soil lead contamination and children's blood lead levels

Howard Mielke of Tulane University and colleagues have analysed the relationship between PbB and soil Pb in Louisiana (Mielke et al., 1997, 2007). The PbB response of children to soil Pb is curvilinear in New Orleans, Louisiana (Mielke et al., 1997, 2007). Johnson and Bretsch (2002) also observed a similar curvilinear relationship between soil Pb and children's PbB in Syracuse, New York. The most recent New Orleans urban soil Pb and PbB study shows the following results: below 100 mg/kg soil Pb children's PbB response is steep at 1.4 μg/dL per 100 mg/kg, while above 300 mg/kg soil Pb children's PbB response is a more gradual 0.32 μg/dL per 100 mg/kg (see Fig. 3; Mielke et al., 2007). It may be hypothesised that similar soil Pb and PbB responses of children are expected in all urbanized areas because the physiological response to exposure is broadly uniform. However, in the USA and Australia, data have shown that African Americans (Lanphear et al., 1996) and Aboriginals (Queensland Health, 2008) tend to have higher PbB than Caucasian children.

The relationship between soil Pb concentrations and blood Pb concentrations have also been modelled using the United States Environmental Protection Agency Mechanistic Exposure Uptake Biokinetic Model for Pb in Children (IEUBK) model (USEPA, 2010). Gulson used the IEUBK model to predict PbB concentrations for a range of children's ages and soil Pb concentrations (Fig. 4; Davis and Gulson, 2005).

The IEUBK soil – PbB slope (Fig. 4) predicts a child PbB level of between 4 and 5 μg/dL following exposure to soil with a Pb concentration exceeding the 300 mg/kg NEPC guideline. Mielke et al.'s (2007) empirical soil and PbB relationship slopes (Fig. 3) predict a PbB level of between 5 and 9 μg/dL for an exposure to soil with a concentration exceeding the 300 mg/kg NEPC (1999) guideline. It is noted that the slope for Mielke et al.'s (2007) empirical model is steeper than the IEUBK model for the first 300 mg/kg.

Soil Pb can also be associated with PbB concentrations greater than 10 μg/dl at soil Pb concentrations lower than suggested by the IEUBK model or Mielke et al.'s (2007) soil Pb–PbB curves. For example, Malcoe et al. (2002) found that logistic regression of yard soil Pb >165.3 mg/kg (OR, 4.1; CI, 1.3-12.4) were associated independently with PbB's greater than or equal to 10 μg/dL. Similarly, the Texas Department of Health (2004), using a large database from El Paso, Texas Area, found an odds ratio 4.5 (1.4, 14.2) for the relationship between a 500 mg/kg increase in soil Pb above background level and childhood blood lead levels > 10 μg/dL.

Laidlaw and Filippelli (2008) performed a review of multiple study designs used to analyse the association between soil Pb and PbB. The study designs included cross-sectional, ecological spatial, ecological temporal, prospective soil removal, and isotopic studies. Sedman (1989) also reviewed multiple American studies published prior to 1989 that demonstrated an association between soil Pb and PbB. In both of these reviews and examples it was shown that PbB in the various studies examined was associated with soil Pb.

The link between soil Pb and PbB was demonstrated recently in New Orleans, where sediments in floodwater from Hurricanes Katrina and Rita (HKR) were deposited onto Pb contaminated soils (Zahran et al., 2010). High density soil surveying conducted

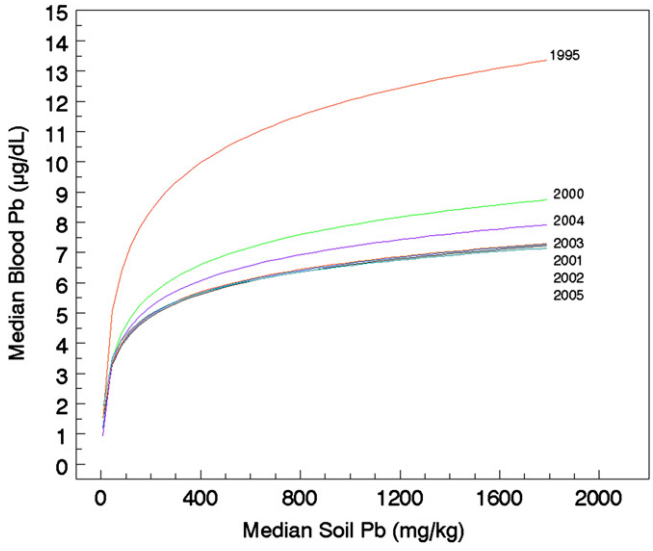

Fig. 3. Median Soil Pb and PbB Curves in New Orleans, Louisiana – 1995 and 2000–2005 (Mielke et al., 2007).

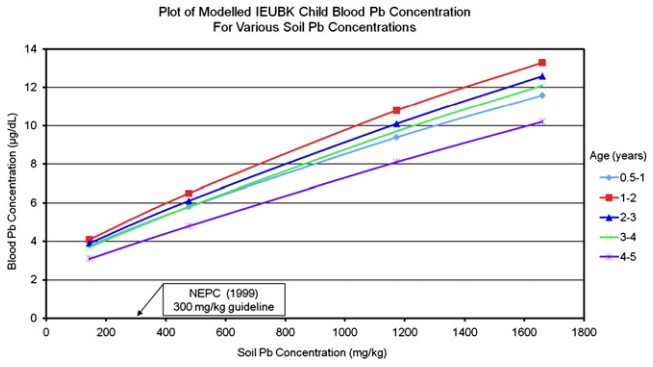

Fig. 4. IEUBK model of children's PbB concentrations give a full range of soil lead concentrations (modified from Davis and Gulson, 2005). This chart shows the IEUBK model of expected changes PbB concentration for a given soil Pb concentration for various age groups of children.

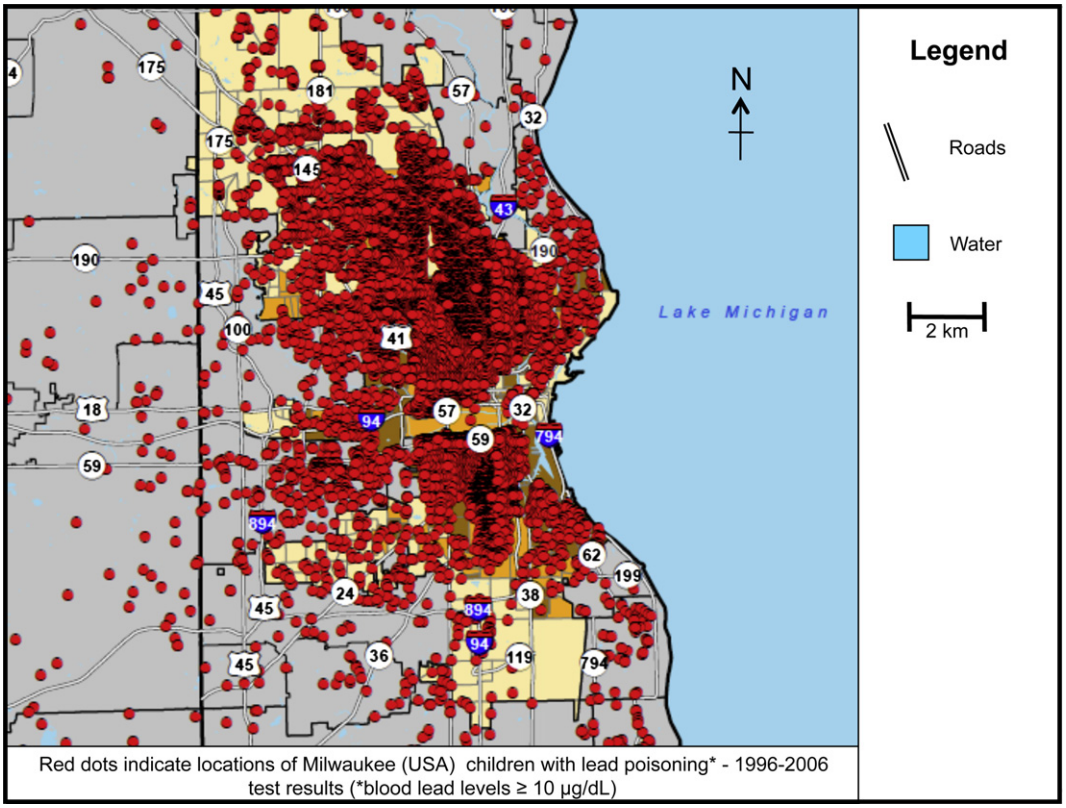

Fig. 5. Milwaukee, Wisconsin (USA) Pb Poisoning Map: 1996–2006. This map depicts the 36,856 children that exhibited PbB poisoning > 10 µg/dL in Milwaukee between 1996 and 2006 (source: modified from the Wisconsin Department of Health, 2010a).

in 46 census tracts before HKR was repeated after the flood. Paired *t* test results show that soil lead decreased from 328.54 to 203.33 mg/kg post-HKR ($t = 3.296$, $p \leq 0.01$). Decreases in soil Pb are associated with declines in children's PbB response ($r = 0.308$, $p \leq 0.05$). Zahran et al. (2010) found that declines in median PbB were largest in census tracts with ≥50% decrease in soil Pb.

Multiple studies in Australia have also shown an association between soil Pb concentrations and PbB concentrations. In Sydney, Fett et al. (1992) found that blood lead concentrations were correlated significantly with concentrations of Pb in yard soil ($r = 0.555$, $p = 0.026$) and play area soil ($r = 0.492$, $p = 0.016$). Young et al. (1992) also observed that soil Pb levels were significantly correlated with PbB levels near the Southern Copper smelter near Wollongong and Bellambi in NSW, Australia. In North Lake Macquarie, NSW near the former Pasminco smelter (Boolaroo, NSW), Willmore et al. (2006) observed that geometric mean PbB was statistically significantly higher for residential soil Pb concentrations greater than 300 mg/kg. In a large study in Sydney, Gulson et al. (2006) observed that the Pb concentration in soil was a significant predictor for Pb in the house dustfall, and dustfall was a significant predictor of PbB. Dustfall accumulation was also observed to be a significant predictor for Pb concentration in handwipes.

6. Australia and United States – PbB screening

Universal blood Pb screening is not performed in Australia (NHMRC, 2009). In 1993, the National Health and Medical Research Council (NHMRC) stated that its specific goal was to achieve for all Australians a blood Pb level of below 10 µg/dL (NHMRC, 1993). This document also recommended a graduated response to PbB levels for both individuals (children of all ages over 15 µg/dL) and communities where >95% of one-to-four-year-old children were below 25 µg/dL, but >5% were above 15 µg/dL. This guideline was rescinded in 2005. In 2009, the NHMRC published an information paper titled *Blood Lead Levels for Australians* (NHMRC, 2009). The document once again supported a 10 µg/dL PbB level guideline and suggested that representative samples of children aged 1–4 living in high and low Pb exposure areas should be screened for PbB. However, while such a blood lead study has been recommended, this has not been undertaken in the inner cities of Australia to date. Currently, due to the lack of universal screening, it is not known what the spatial distributions and incidence levels are of children with elevated PbB levels. Therefore it is difficult to establish the exact nature of the risk in urban city areas.

In the US, the current PbB screening practices were described by Cole and Windsor (2010), who stated: "*….lead screening practices are created at the state level, with each state identifying and agreeing on its own lead screening guidelines. States vary widely in their approach to lead screening. Most states have a plan targeting children under the age of 6, but these plans vary greatly. Some states advocate universal screening (ex. Tennessee, Connecticut) while some advocate risk-based screening (ex. Illinois, Florida). Risk-based screening is usually accomplished through a parent questionnaire that identifies children who may be at higher risk for lead exposure and then only testing those at-risk. In addition, some states test children of certain SES designations, or who live in lower income areas or in older housing.*"

7. Australian and United States blood lead prevalence

In comparison to PbB studies in mining towns (see Taylor et al., 2010), there have been few PbB prevalence studies completed in

inner city areas of Australia. Fett et al. (1992) determined the distribution of PbB levels in 158 preschool children in inner Sydney and observed that 50.6 % of the children had PbB levels > 10 μg/dL, 17.1 % had PbB levels > 15 μg/dL, and 2.5 % had PbB's > 25 μg/dL. In a PbB survey of 718 children in central and southern Sydney, Mira et al. (1996) observed that 25 % of the children had PbB > 10 μg/dL and 7 % had PbB > 15 μg/dL. The only nationwide survey of PbB concentrations was conducted on 1575 children in 1995 (Donovan, 1996). Donavan found that the geometric mean PbB concentration in 1995 in 1–4 year-olds was 5.1 μg/dL, with 7.3 % exceeding 10 μg/dL and 1.7 % exceeding 15 μg/dL. A PbB prevalence study in Fremantle (Willis et al., 1995) of 120 children from day-care centres and 44 hospital inpatients observed that 25.6 % had PbB's > 10 μg/dL. A recent five-year longitudinal study of 113 children living in Sydney, aged six months to 31 months at recruitment, showed a mean PbB concentration of 3.1 μg/dL (range = 0.6–19.0 μg/dL) (Gulson et al., 2006). A PbB survey of 100 participants in Fremantle Western Australia in 2008 (Guttinger et al., 2008) found that none had PbB's ≥10 μg/dL. It is likely that PbB levels in Fremantle (Willis et al., 1995; Guttinger et al., 2008) have declined due to the elimination of Pb in petrol. However, it must also be noted that PbB prevalence studies with low sample numbers of around 100–150 subjects, as was done in Fremantle by Guttinger (2008) and in Sydney by Gulson et al. (2006), are not likely representative of the geographic distribution of PbB levels of large populations. For example, in Mt. Isa, Australia, Queensland Health determined that a sample size of 400 (approximately 25% of the Mount Isa population of children aged one to four) was required to have sufficient power to provide reliable information on PbB levels (Queensland Health, 2008). A city the size of Sydney would require a much larger sample to be statistically significant compared to Mount Isa.

The United States Center for Disease Control (CDC) indicates that the prevalence of PbB < 10 μg/dL in the US during 1999–2002 survey period for children aged 1–5 years was 1.6 % (CDC, 2005). However, the national results are arguably misleading because of the emerging evidence of the effects of low levels Pb exposure (Canfield et al., 2003; Schnaas et al., 2006; Surkan et al., 2007; Chiodo et al., 2007; Lanphear et al., 2005; Miranda et al., 2007; Chandramouli et al., 2009; Zahran et al., 2009; Nigg et al., 2010). The National Health and Nutrition Examination Survey (NHANES) III 1999–2002 database indicates that approximately 2.4 million children aged 1–5yrs old have PbB levels between 5 and 9.9 μg/dL (Iqbal et al., 2008). Within the population sample with blood Pb levels of 5 μg/dL or higher, the prevalence was 47% for non-Hispanic black children, 28% for Mexican American children, and 19% for non-Hispanic white children (Bernard, 2003). Further, the distribution of affected children is highly spatially skewed. The prevalence of PbB poisoning > 10 μg/dL in inner cities of the US exceeds 10 to 20% in many cities. For example, the city of Milwaukee, Wisconsin, which has a population of approximately 1.7 million, has soils in the central city area contaminated with Pb (mean = 640 mg/kg, median = 280 mg/kg) (Brinkmann, 1994). Milwaukee's childhood PbB levels peak in the summer and early autumn and have been correlated to particulate matter less than 2.5 μm (Havlena et al., 2009). The seasonal variation of PbB was hypothesised by Havlena et al. (2009) to be related to the availability of dust and airborne particulates during summer months. Fig. 5 shows the distribution of Milwaukee's 36,856 children with PbB poisoning (i.e. >10 μg/dL) between 1996 and 2006. In 2008 the citywide prevalence rate for PbB >10 μg/dL was 4.8 %, but was much higher in some neighbourhoods as indicated on a PbB incidence map on Fig. 5 (after Wisconsin Department of Health, 2010a). This demonstrates that while average PbB levels may be relatively low, the incidence of PbB poisoning exceeding 10 μg/dL (or even 5 μg/dL) may be elevated and represent large numbers of children in the inner-cities. It is notable that the soil Pb concentrations in some of the inner Sydney suburbs are higher than those in Milwaukee. This might suggest at least an equal or greater risk than that which has already been demonstrated to exist in Milwaukee.

8. Toxicity of low level Pb exposure typically caused by exposure to Pb in soil dust

The current Pb guideline in Australia is a PbB concentration of 10 μg/dL (NHMRC, 2009). However, emerging evidence (see below) suggests that the definition of Pb poisoning in Australia may need to be reduced to 5 μg/dL, or even lower at 2 μg/dL (Taylor et al., 2010). In Australia, this could result in the emergence of a large number of Pb poisoned children. Low PbB levels (<10 ug/dL) typically associated with urban soil Pb exposure are associated with a myriad of health outcomes. Low PbB levels (<10 ug/dL) are associated with Attention-Deficit Hyperactivity Disorder (ADHD) (Nigg et al., 2010), a reduction in children's tests scores for reading (odds ratio = 0.51, $p = 0.006$) (Chandramouli et al., 2009), writing (odds ratio = 0.49, $p = 0.003$) (Chandramouli et al., 2009; Miranda et al., 2007) and mathematics (Miranda et al., 2007). Canfield et al. (2003) observed that when lifetime average PbB concentrations in children increased from 1 to 10 μg/dL, the intelligence quotient (IQ) declined by 7.4 points. Jusko et al. (2008) observed that compared with children who had lifetime average PbB concentrations < 5 μg/dL, children with lifetime average concentrations between 5 and 9.9 μg/dL scored 4.9 points lower on Full-Scale IQ (91.3 vs. 86.4, $p = 0.03$). Similarly, Surkan et al. (2007) observed that children with 5–10 μg/dL had 5.0 (S.D. 2.3) points lower IQ scores compared to children with PbB levels of 1–2 μg/dL ($p = 0.03$). Interestingly, multiple studies have shown that that the strongest Pb effects on IQ occurred within the first few micrograms of PbB (Schnaas et al., 2006; Canfield et al., 2003; Lanphear et al., 2005). Low PbB levels (<10 ug/dL) have also been associated with various physiological outcomes such as kidney damage (Fadrowski et al., 2010), dental caries (Moss et al., 1999), puberty delay in boys (Williams et al., 2010) and girls (Selevan et al., 2003) and cardiovascular outcomes in adults (Navas-Acien et al., 2007).

9. Urban soil lead exposure prevention

Although the currently acceptable soil Pb guideline for residential housing is 300 mg/kg Australian, (NEPC, 1999), in Norway the soil Pb guideline for children's play areas is 100 mg/kg, and in California the draft soil Pb guideline is 80 mg/kg (California, 2009). Currently, a full-scale national program is underway in Norway to reduce soil Pb values below 100 mg/kg at all childcare centres, elementary schools and parks in the 10 largest cities (Ottesen et al., 2008). The driver of this cleanup was that in 2005 the Norwegian government promised that every child between the age of 1 and 6 years should have access to daily care in day-care centres, if desired by their parents. As a result, many new day-care centres were established, especially in the cities, and about 75% of all children in this age group spend 6–9 hours in such centres on work-days. Studies of soil pollution in day-care centres in Norway's three largest cities, Trondheim, Bergen and Oslo between 1996 and 2007 revealed the need for soil remediation (including Pb) in up to 38 % of locations (Ottesen et al., 2008). Given the known relationship between environmental Pb and traffic and its impacts on adjacent soils and dusts (LaBelle et al., 1987; Laidlaw and Filippelli, 2008), it is quite probable that schools and day care centres on high traffic volume streets in Sydney and other larger Australian cities may also require remediation similar to that conducted in Norway.

In Australia, precedent for the remediation/isolation of urban soils has been set in Boolaroo NSW near the location of the former Pasminco smelter. The NSW Department of Environment and Climate has approved a soil Pb abatement strategy for approximately 4000 properties surrounding the former Pasminco smelter at Boolaroo (Lake Macquarie Council, 2009). Soils with Pb concentration ranging between 300 mg/kg and 2500 mg/kg will be covered with clean soil and areas where soil concentrations exceed 2500 mg/kg the top 5 cm of soil will be removed. In the US, Mielke et al. (2006) demonstrated that exposure to Pb contaminated urban soils can be prevented by covering contaminated soils with about 15 cm of low Pb (median ~5 mg/kg) soil. To achieve that, clean soil is simply graded over the old soil layer, hydroseeded (a slurry of seeds and moisture-retaining fill mixture sprayed onto the ground), and left to grow into a lawn. This approach "caps" the Pb-contaminated soils, and prevents children from coming into contact with soil-borne metals. Yard remediation has been demonstrated to effectively reduce PbB levels. For example, Maisonet et al. (1997) found that yard soil remediation was a protective factor for elevated PbB levels in children (odds ratio, 0.28; confidence interval, 0.08–0.92).

10. General comparisons – Australia and the United States

Differences in climate, urban space and socio-economic, ethnic, and racial make-ups may affect exposure to Pb in soil dust in the US and Australia. The climates in the two counties are highly diverse geographically. Sydney has a climate similar to San Diego California, but with more precipitation, Melbourne is located in a climate similar to North Carolina, Adelaide and Perth have similar climates to Los Angeles or San Francisco and Brisbane has a climate similar to the state of Florida. It appears that the garden or lawn spaces in the US inner city areas are larger than in Australian inner cities due a higher building density in Australian cities. Some areas of Australian older inner cities have minimal garden or lawn spaces. This may reduce exposures to lead in soil dust compared to US inner cities. The socioeconomic makeup in the inner cities in the US is also different. In Australia, the inner city residents are comprised primarily of Caucasian, Asian and other ethnic groups with relatively higher socioeconomic status than their US counterparts. In the US, large portions of the inner cities are of lower socioeconomic status consisting of African American, Latino and lesser numbers of Caucasians. The behaviours of people of different socioeconomic levels and racial groups could result in different exposure patterns. Furthermore it is possible that the contribution to PbB caused by exposure to interior Pb paint may be different between the countries due to different physical properties of the paint.

11. Blood lead prevalence

It is not possible to quantify the number of children with blood lead poisoning in Australian inner cities because no recent statistically adequate blood lead prevalence studies have been conducted. The soil lead concentrations in Sydney, presented above, may be roughly similar to the soil lead concentrations in New Orleans or Milwaukee. The blood lead prevalence in New Orleans in 2005 was 10.7 % for PbB > 10 μg/dL and 94.1 % for PbB > 2 μg/dl (Howard Mielke, personal communication). In Milwaukee the citywide prevalence for PbB > 10 μg/dL was 4.8 % in 2008. However, PbB prevalence rates > 10 μg/dL are insufficient indicator of risk due to the clear toxicity of PbB concentrations > 5 μg/dL. The 2002 NHANES data indicated that in the US, that for every case of PbB >10 μg/dL (CDC, 2005), there were approximately 7.7 PbB cases between 5 and 9.9 μg/dL (Iqbal et al., 2008).

12. Summary

A review of the scientific literature from Australia and the US indicates that some of the inner-city soils in both countries are variously contaminated with Pb and that soil Pb correlates with children's PbB levels. However, unlike the US, the spatial and temporal pattern of children's PbB levels in Australian inner-city children remains poorly characterised, with the exception of a few limited non-systematic studies. Similarly, the soil Pb distribution in large and regional Australian cities is also characterised by ad-hoc non-systematic studies. Therefore, it is argued here that the risks from low-level Pb exposure from urban soils, the likely predominant Pb reservoir, are unconstrained. Consequently, it is not possible to determine what the health risks are or what appropriate prevention strategies ought to be, although the data point to the potential for a high prevalence of PbB poisoning (>5 ug/dL) in some older inner-city areas of Australia's major cities.

Lead concentrations in some inner-city Sydney areas indicate that soils are highly contaminated and approach the soil Pb concentrations near the former Pb and zinc smelter in North Lake Macquarie (Willmore et al., 2006). We suggest that there is an urgent need for high density soil Pb mapping and universal PbB screening in older areas of large Australian cities with a history of high traffic volumes. Widespread soil Pb remediation should also be evaluated as a method of preventing children's exposure to soil and dust containing Pb in Australia's large inner cities. This is necessary if Australia is to take a precautionary approach to the risks of environmental Pb exposure (Taylor et al., 2010).

13. Recommendations

1) High density soil Pb mapping should be performed in the inner cities of Australia (see Mielke, 1991, 1994; Mielke et al., 2005);
2) On completion of soil Pb mapping in large Australian inner cities, we recommend that an initial PbB screening be targeted in areas where soil Pb concentrations exceed the 300 mg/kg guideline. The PbB screening should be sampled during the summertime because PbB is known to be highest during the summertime (Laidlaw et al., 2005; Laidlaw and Filippelli, 2008; Laidlaw, 2010). Following the initial PbB screening, targeted screening should be terminated in areas that exhibit a low percentage of PbB > 5 μg/dL. However in areas with high percentages of children with PbB > 5 μg/dL, PbB screening should continue until the Pb source or sources are remediated and PbB levels reduced below 5 μg/dL for at least 95% of the children. A PbB concentration of >5 μg/dL was used as the intervention PbB level in Esperance (Western Australia Government Committee of Inquiry Education and Health Standing Committee, 2007). This has recently (2007) become the default action level for children < 5 years old for Western Australia. In addition to remediating soil sources in these areas, it would also be prudent to seal indoor and outdoor flaking Pb paint to prevent further interior particle contamination and exterior soil contamination (Gulson et al., 1995); and
3) For transparency, we recommend that all PbB cases > 5 μg/dL be plotted on a GIS map of each city and be made available on the Internet as has already been done by the Wisconsin Department of Health (2010b). In addition, the proposed high density soil lead maps of the large Australian cities should be placed in the same location on the internet. This will allow residents to monitor evidence of progress in the elimination of children's PbB levels and will allow current and future residents to make an informed choice about any potential risks with respect to choices of homes and schools.

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Chapter 2 - Paper 2: "*Identification of lead sources in residential environments: Sydney, Australia."*

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Identification of lead sources in residential environments: Sydney Australia

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ABSTRACT

Interior and exterior dust, soil and paint were analysed at five brick urban Sydney homes over 15 months to evaluate temporal variations and discriminate sources of lead (Pb) exposure. Exterior dust gauge Pb loading rates (μg/m^2/28 days), interior vacuum dust Pb concentrations (mg/kg) and interior petri-dish Pb loading rates (μg/m^2/28 days), were correlated positively with soil Pb concentrations. Exterior dust gauge Pb loading rates and interior vacuum dust Pb concentrations peaked in the summer. Lead isotope and Pb speciation (XAS) were analysed in soil and vacuum dust samples from three of the five houses that had elevated Pb concentrations. Results show that the source of interior dust lead was primarily from soil in two of the three houses and from soil and Pb paint in the third home. IEUBK child blood Pb modelling predicts that children's blood Pb levels could exceed 5 μg/dL in two of the five houses.

1. Introduction

In many urban inner-city areas in the United States, there is an epidemic of childhood PbB poisoning (Gould, 2009). It has been estimated that 24.5%, or 9.6 million US children have a PbB in the 2–10 μg/dL range, a level which will cause sub-clinical signs (Gould, 2009). The United States Centers for Disease Control and Prevention (CDC) estimates that in the United States approximately 535,000 children aged 1–5 years had BLLs ≥5 μg/dL (CDC, 2013). These exposures are quite variably geographical with some locations more significantly affected. For example, in New Orleans children currently have a PbB prevalence (>5 μg/dL) of 29.6% (Mielke et al., 2013) and Detroit children (aged 0–10 years) have a PbB prevalence of 33% (>5 μg/dL) (Zahran et al., 2013). In 2012, the United States CDC Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP, 2012) recommended the adoption of a children's PbB reference level of 5 μg/dL. While the PbB prevalence has been assessed in the United States, in Australia childhood PbB surveillance are not collected and reported systematically so spatial and temporal distributions are unknown. The last national PbB testing occurred in 1995, when 1575 children were tested (Donovan, 1996). The arithmetic mean PbB level was 5.72 ± 3.13 μg/dL. Currently, PbB testing programs are focussed on Australia's Pb mining and smelting towns: Broken Hill, Mount Isa and Port Pirie (Taylor et al., 2011). Similar to the United States, there is no federal government program for testing or remediation of diffuse non-point source urban soil Pb contamination, although the extent of these sources is increasingly better understood, particularly in urban neighbourhoods (Olszowy et al., 1995; Birch et al., 2011; Laidlaw and Taylor, 2011).

The premise of this study is derived from Laidlaw and Taylor's (2011) review of multiple Australian soil Pb and dust Pb studies that concluded soils and interior dust in many older Sydney suburbs are likely to have been contaminated from industrial and domestic Pb sources. In support of this contention, is the work by Birch et al. (2011) who mapped soil Pb concentrations in the Sydney basin and observed widespread soil Pb contamination with highest concentrations located in the inner parts of eastern, northern and western Sydney.

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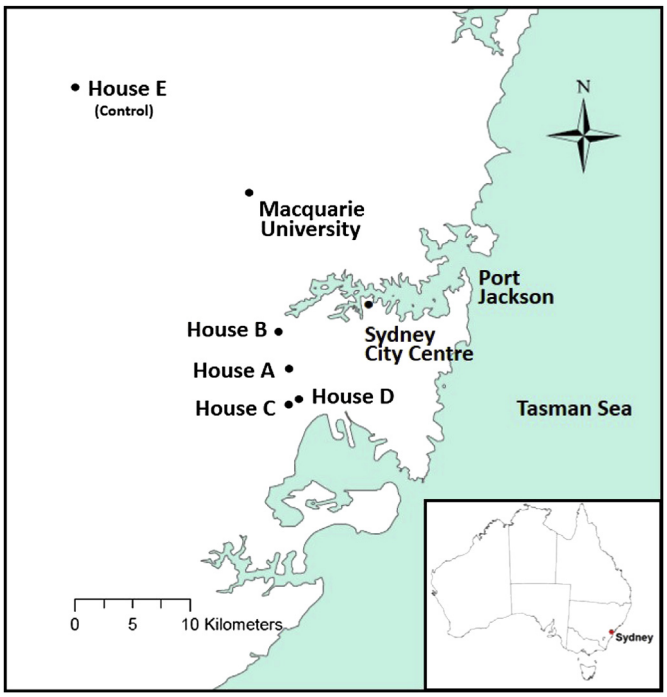

Fig. 1. Site locations of houses sampled in the study. This figure displays the locations of the houses that participated in this study.

This study examines sources of residential Pb and its temporal cycling in 5 brick homes over a 15 month period during 2010–2012 in Australia's largest urban city, Sydney (population 4.61 million; Australian Bureau of Statistics (ABS), 2012). The principal aim of the study was to determine the predominant source(s) of Pb inside typical western Sydney brick homes. Brick homes were selected to avoid the potential confounding effect of exterior lead paint that was used in older homes. Given that the study involved the collection of longitudinal environmental data, it was also possible to evaluate the seasonal variation in exterior and interior Pb quantities.

2. Methods and approach

2.1. House selection

The criteria for the four principal study houses located in the older inner west of Sydney was that each house was greater than 50 years old and was constructed of unpainted bricks. The four houses were built between 1900 and 1918. The houses were selected from three inner west Sydney suburbs: Arnfield (2), Marrickville and Haberfield (Fig. 1). A fifth brick house, approximately 30 years old was also sampled as a reference site. This property was located in an area of low-density bushland in the suburb of Glenhaven, 28 km northwest of Sydney city centre (Fig. 1). The owners of each home gave their consent to environmental sampling.

2.2. Study design

The sampling period was undertaken between November 2010 and January 2012 (15 months). At each house, exterior atmospheric Pb loading rates were measured monthly using passive sampling dust gauges in the rear garden at each home. Interior vacuum Pb in the <75 μm fraction were analysed monthly and attic and interior petri-dish Pb loading rates were measured quarterly. Soil Pb concentrations (<75 μm fraction) were measured in surface soil samples (0–2 cm) collected adjacent to the roadway in front of each house, within 1 m from the front of each house and in the middle of each back yard. In addition, one soil sample was collected at a depth of 50 cm in the middle of each back yard. Using the three inner west homes with the highest total soil Pb concentrations (Houses A, C and D), Pb isotopic composition analysis of selected environmental samples was undertaken using quadrupole Inductively Coupled Mass Spectrometry (ICP-MS). These samples consisted of front yard surface (0–2 cm) and sub-surface (50 cm) soils (<75 μm fraction); a vacuum dust sample (<75 μm fraction); and an indoor paint chip sample from the surface identified previously to have the highest Pb concentration. Finally, Pb speciation of vacuum dust and surface soil (0–2 cm) samples were determined from the same three houses (Houses A, C and D) using X-Ray Absorption Spectroscopy (XAS) at the Australian Synchrotron facility in Melbourne, Australia.

2.3. Sieving

Soil and vacuum dust samples were sieved using a 75 μm mesh prior to analyses. Sieves were rinsed in a tap water/Alconox™ solution followed by rinsing with type II deionised water (American Society of Testing Materials (ASTM) standard) and then dried at 85° C before and after use. Soil and dust samples selected for XAS analysis were milled to <20 μm with a Retsch MM301 tungsten carbide milling machine in order to mitigate sample thickness effects that could otherwise distort the spectra. The mills were cleaned with fine silica sand followed by Alconox wash and type II deionised water rinse.

2.4. Soil and vacuum samples

Each surface soil sample was a composite of three samples collected approximately 1 m apart using a plastic hand trowel that was cleaned with deionised water and dried between sample locations. Samples were collected and stored in metal-free plastic bags prior to sieving.

Monthly interior vacuum dust samples (1 sample per location per month) were collected from each house over the study period, all of which used High Efficiency Particulate Air (HEPA) vacuum cleaners. Four of the vacuum cleaners (Houses A, C, D and E) were bag-less and one contained a vacuum bag (House B). The entire content of each vacuum sample was placed in a large, resealable metal-free plastic bag. Sieved (<75 μm) soil and vacuum samples were analysed for total extractable Pb concentrations using United States Environmental Protection Agency (USEPA) SW-846-6010 (USEPA, 2013b) method by the ALS Laboratory Group in Sydney, Australia.

2.5. Dust gauge samples

Exterior dust gauges were placed in the rear garden areas of each house in a location where they would not be disturbed. The dust gauge consisted of a 150 mm diameter glass funnel which was inserted into a 2.75 L glass bottle secured in a plastic bucket affixed to a ~2 m high tripod (Australian Standard 3580.10.1-2003; Standards Australia, 2003). Dust gauge bottles were replaced monthly over the study period (1 sample per location per month). Ten ml of copper sulphate solution was inserted into each bottle to prevent algal growth. Dust gauge samples were analysed for total Pb concentrations measured using NexION 300D ICP-MS by the ALS Laboratory Group in Sydney. Total extractable Pb concentration was analysed using USEPA Method SW-846-6020 (USEPA, 2013b).

2.6. Petri-Dish samples

Petri-dish(es) (150 mm or 85 mm diameter) were placed in the attic of each house and on the main living area of each house at approximately 2 m above the floor to collect settled airborne interior dust. The polycarbonate petri-dishes were purchased in an air-tight plastic and were pre-sterilised. Petri-dishes were replaced on a quarterly basis during the study (five quarters over the study period). The main living area petri-dish was placed in the baby's bedroom in house D, and in the living room/family room of houses A, C, B and E. Each petri-dish sample was analysed for the mass of Pb using ICP-MS by the ALS Laboratory Group in Sydney. Samples were digested using a concentration of 7 M nitric acid and 6 M hydrochloric acid according to the method described in Wlodarczyk et al. (1997). Interior and attic petri-dish lead dust loading rate samples were collected quarterly (1 sample per location every three months).

2.7. Paint chip analysis

Paint chip samples were collected from the interior paint inside each house using new disposable razor-blades. Samples were stored in metal free plastic freezer bags prior to analysis for total Pb concentration. Paint chips with the highest concentration in houses A, C and D were analysed for their Pb isotopic composition.

2.8. Pb isotopic composition analysis

Total Pb paint concentrations and Pb isotopes in soil (sieved < 75 μm), vacuum dust (sieved < 75 μm) and Pb paint were analysed using a quadrupole Inductively Coupled Plasma-Mass Spectrometry (ICP-MS) at the ChemCentre in Bentley, Western Australia. In addition, several paint samples used for initial screening were also analysed for total Pb concentrations at ALS Laboratories. Soil and dust particles (sieved < 75 μm) were digested after drying overnight. Analysis was performed using mixed high purity redistilled acid (nitric/hydrochloric) microwave assisted acid digestion (USEPA 3051A modification). Paint samples were digested with strong reflux with nitric acid as per the Association of Official Agricultural Chemists (AOAC) method 974.02 (AOAC, 2013). Sample digests were filtered and volumed in 18 MOhm water before determination of total Pb content by ICP-AES. Samples measured for their Pb isotopic composition were diluted within a concentration range of 10–

Table 1
Descriptive summary statistics of Pb by house across various sampling locations. This table contains the average soil, vacuum dust Pb, exterior dust gauge, attic petri-dish, house petri-dish Pb concentrations for houses A through E.

	House A Marrickville, Parade street	House B Haberfield, Rogers street	House C Arncliffe, Forest road	House D Arncliffe, west Botany street	House E Glenhaven, Bannerman road
Soil (mg/kg)	714 [$n = 3$] (17)	261 [$n = 3$] (79)	1203 [$n = 3$] (482)	451 [$n = 3$] (287)	47 [$n = 3$] (10)
Vacuum dust (mg/kg)	575 [$n = 13$] (240)	265 [$n = 12$] (177)	1159 [$n = 15$] (787)	256 [14] (54)	189 [6] (49)
Exterior dust gauge (Pb μg/m^2/28 days)	257 [$n = 15$] (198)	126 [$n = 15$] (84)	248 [$n = 15$] (162)	187 [$n = 15$] (208)	35 [$n = 7$] (62)
Petri-dish attic (Pb μg/m^2/28 days)	44 [$n = 5$] (42)	2 [$n = 5$] (5)	275 [$n = 5$] (20)	67 [$n = 5$] (60)	9 [$n = 2$] (17)
Petri-dish house (Pb μg/m^2/28 days)	11[$n = 5$] (13)	<2 [$n = 5$] (0)	25 [$n = 5$] (20)	10 [$n = 5$] (16)	<2 [$n = 1$] (0)
Paint (mg/kg)	1723 [$n = 3$] (1260)	7910 [$n = 1$] (0)	13,748 [$n = 3$] (13,379)	1750 [$n = 2$] (717)	NA

Note: Standard deviation in parentheses; *n* in brackets; *Values represent averages of samples collected from front street, front house side and rear yard. Vacuum Bag Dust and Exterior Dust Gauge – sampled monthly; Petri-Dish Samples – sampled quarterly.

25 μg/L in 0.1% HNO_3 to remain in pulse detector counting mode of the instrument, confirmed for concentration, and then run for Pb isotopic ratios using a pass stable sample introduction (SSI) peltier double cooled spray chamber after optimisation on an Agilent 7500 ICP-MS. Each sample was bracketed before and after by a mass bias correction against the certified reference material National Institutes of Standards and Technology (NIST)-981. In addition, certified reference material NIST-982 was run after every 20 samples as an external quality control. Acquisition was for a total across the isotopes of 24 s for each reading and 5 readings were then averaged for the final result.

2.9. X-ray Absorption Spectroscopy (XAS)

Extended X-Ray Absorption Fine Structure (EXAFS) measurements were carried out at XAS beamline 12ID at the Australian Synchrotron (Melbourne, Australia). Samples were diluted in microgranular cellulose (Sigma–Aldrich) and pressed into pellets of 1 mm thickness using a hydraulic press. The Pb LIII-edge was measured in fluorescence mode with a 100-element solid-state Ge detector at 90° to the incident beam, with 45° sample geometry. Energy was scanned using a liquid nitrogen cooled Si (111) double crystal monochromator, and the energy scale was calibrated on the Pb LIII edge using a Pb standard foil. Samples were measured using energy stepping of 10 eV for the pre-edge baseline, 0.25 eV through the XANES region and steps of 0.035 *k* in the EXAFS region. Harmonics were suppressed by the use of a collimating mirror upstream of the monochromator, and a sagitally focussing mirror downstream to the monochromator, both of which were Rh coated yielding an energy cut-off of ~18 keV. During collection the storage ring was operated at 3 GeV with ring current in the range 150–200 mA. At least two spectra for each sample were collected; spectral averaging and dead time correction were carried out using Australian Synchrotron software.

2.10. Quality assurance procedures

Three of soil and three vacuum dust samples were split in the field, each sample homogenized, and sent to the laboratory for analysis.

Table 2
Least squares and least absolute deviation regression coefficients predicting Pb content of exterior dust gauge as a function of soil Pb content. This table presents the statistical results of least squares regressions predicting Pb content of exterior dust gauge as a function of soil Pb content.

	LAD gauge	LAD gauge 3 month moving average	OLS *ln* gauge	OLS *ln* gauge 3 month moving average
Soil Pb	0.067*	0.076**		
	(0.039)	(0.034)		
ln soil Pb			1.724***	1.530***
			(0.464)	(0.452)
Winter (Reference)				
Autumn	−7.228	76.668*	−2.316	3.710***
	(48.43)	(39.23)	(1.479)	(1.279)
Spring	50.337	47.568	2.655**	4.005***
	(43.26)	(37.45)	(1.315)	(1.238)
Summer	157.34***	139.12***	5.451***	6.579***
	(42.00)	(35.88)	(1.278)	(1.176)
Constant	−22.574	−34.495	−12.42***	−11.06***
	(39.64)	(35.50)	(2.933)	(3.004)
Observations	67	57	67	57
F-test			12.01	9.79
Pseudo *R*-squared	0.274	0.333		
R-squared			0.450	0.430

LAD = least absolute deviation regression; OLS = ordinary least squares regression.
Note: Standard errors in parentheses, ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$.

Out of the seven Pb field duplicate Relative Percent Difference (RPD) pairs (3 soil, 3 vacuum dust and 1 paint) reported, all the RPDs were less than 3%, with the exception of paint, which had an RPD of 12.5%. Petri-dish blanks were below instrument detection while dust gauge field blanks returned Pb values of (0.002 mg/L), close to the instrument detection limit (0.001 mg/L). Blank values were subtracted from the dust gauge measurements. The source of the Pb may have originated from the copper sulphate solution that was added to the bottles to prevent biological build-up (ALS Laboratory Group, personal communication). Overall, the results of the RPD calculations between primary and duplicate samples indicate that the analytical data are suitable for interpretive use. Laboratory quality assurance procedures included analysis of matrix spike/matrix spike duplicate (MS/MSD) results data, method blank data, laboratory control spike data, regular sample surrogate data and laboratory duplicate data. The overall results of the laboratory quality assurance are suitable for interpretive use, i.e. between 75% and 120%. The relative standard deviation percentage (RSD%) for Pb isotope analysis was 0.24, 0.22, 0.21 and 0.23 for the lead ratios of $^{207}Pb/^{206}Pb$, $^{208}Pb/^{206}Pb$, $^{207}Pb/^{204}Pb$ and $^{206}Pb/^{204}Pb$, respectively. Overall, the analytical results are considered suitable.

2.11. IEUBK modelling

The USEPA's Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) software version IEUBKwin1_1 Build11 was used to predict PbB levels in each house (USEPA, 2013a). Average soil Pb and vacuum dust Pb concentrations (Table 1) were used for the soil and indoor dust input variables. The Pb absorption fraction percent used was 23%(46% bioavailable), which is a conservative value based upon Snowdown and Birch's (2004) study of soil bioavailability values in Sydney. An air Pb value of 0.02 μg/m^3 was used in the model, which was based upon the Australian Nuclear Science and Technology Organisation recent air monitoring station data 5 km south of the Sydney CBD (ANSTO, 2013). IEUBK default values were used for the dietary Pb input variables. A value of zero was used for the drinking water contribution given that the most recent data on Sydney's water quality shows values are below detection limits (Sydney Water, 2013).

3. Results

3.1. Lead analysis

Descriptive summary statistics of Pb concentrations in soil, vacuum dust, exterior dust loading, petri-dish (attic and interior) and paint are presented in Table 1. The mean soil Pb concentration at the reference home located in Glenhaven at 47 mg/kg (std. dev. = 10 mg/kg) (House E) is slightly larger than the Sydney soil Pb background level of 16 ± 3.5 mg/kg identified by Birch et al. (2011). The mean of the sample at House E included a roadside soil sample (37 mg/kg), a rear garden sample (56 mg/kg), and a soil sample collected beside the house (49 mg/kg). The soils at this location were present when Pb was still being used as an additive in petrol. House dust Pb concentrations are presented in Supplemental Table S2.

We begin by analysing variation in exterior dust gauge Pb loadings using both *least squares and least absolute deviation regression* procedures (Table 2). Columns 1 and 2 report median regression results. Adjusting for seasonality, Column 1 shows the expected change in the Pb content of exterior dust gauge (at the median of the conditional distribution of exterior dust gauge loadings) per unit (mg/kg) increase in soil Pb. We find that dust gauge Pb loadings increase by 0.066 μg/m^2/28 days for every mg/kg

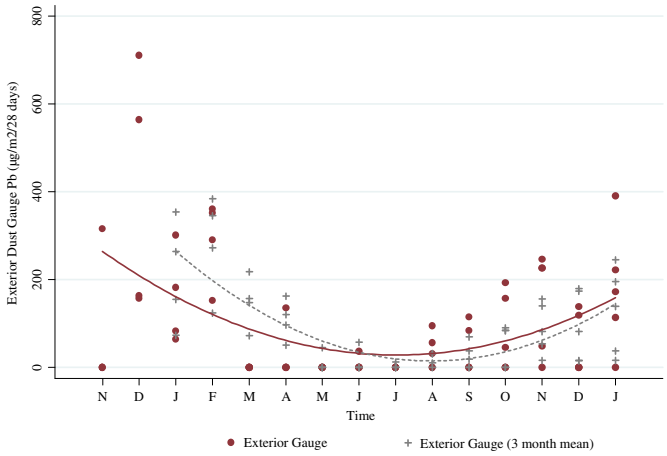

Fig. 2. Exterior Pb dust gauge data at the monthly time-step with fractional polynomial fit. This figure displays monthly variations in atmospheric Pb loading rates (µg/m^2/28 days) collected in atmospheric dust gauges that were placed in the rear yards of the study houses between November 2010 and January 2012. The results indicate that atmospheric Pb loading peaks in the summer/autumn and is lowest during the winter. Red dots correspond to the exterior dust gauge Pb loading reading for each dust gauge for the month of observation. Grey crosses corresponding to the 3 month moving average. The red line intersecting the space corresponds to a best fit quadratic line for red dots. The same logic obtains for the grey dotted line. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

increase in soil Pb. Results, in Column 2, behave near identically if we regress a three-month rolling average of dust gauge Pb loadings on soil Pb ($b = 0.076$, $p < 0.05$). Interestingly, and consistent with a soil re-suspension hypothesis, results show that dust gauge Pb readings are significantly higher in the summer period (Column 1, $b = 157.34$, $p < 0.001$) compared to the reference season of winter gauge readings. The seasonality of exterior Pb loadings is shown in Fig. 2, which reveals dust gauge Pb loadings are lower in the Australian winter months of June to August, and higher in the summer months (December to February). The seasonality of temperature in Sydney follows the same pattern (BOM, 2013). Columns 3 and 4 report least squares regression results. To account for skew, both soil Pb and dust gauge Pb variables are log transformed. By taking the natural log of both sides of the regression equation, allowing us to express the relationship in percentage terms, in Column 3 we find that a 1% increase in soil Pb is associated with 1.72% increase in dust gauge Pb loadings. Results pertaining to the three-month rolling average of dust gauge Pb loadings reported in Column 4 behave similarly.

Given that a substantial fraction of the statistical variation in exterior dust gauge Pb loadings is explained by soil Pb accumulation, the variation in the Pb content of vacuum dust collected from homes over a 15 month period can be determined. Table 3 presents least absolute deviation and least squares regression coefficients predicting the Pb content of vacuum dust as a function of soil Pb, interior paint Pb, and seasonal dummy variables. Table 3 repeats the measurement and analytic logic of Table 2, where monthly, three-month rolling average, and natural log transformations of our response variable are regressed on relevant predictors. In Column 1 of Table 3, we find that each 1 mg/kg increase soil Pb induces a 0.803 mg/kg (95% CI, 0.369–1.238) increase in the median of the distribution of Pb content of vacuum dust. In Column 2, results show that a unit increase soil Pb significantly increases the three-month moving average of the accumulation of Pb in vacuum dust ($b = 1.050$, $p < 0.01$). Interior paint Pb is not statistically associated with Pb vacuum dust in either least absolute deviation regression model. In Columns 3 and 4 results from our log–log least squares

Table 3
Least squares and least absolute deviation predicting Pb content of interior vacuum dust as a function of soil and paint Pb content.** This table presents the statistical results of least squares and least absolute deviation regressions predicting the Pb content of interior vacuum dust as a function of soil and paint Pb content.

	LAD vacuum	LAD vacuum 3 month moving average	OLS *ln* vacuum	OLS *ln* vacuum 3 month moving average
Soil Pb	0.803***	1.050***		
	(0.216)	(0.131)		
Paint Pb	0.024	0.024		
	(0.026)	(0.015)		
ln soil Pb			1.185**	1.847***
			(0.367)	(0.260)
ln paint Pb			0.071	0.248**
			(0.146)	(0.103)
Winter (Reference)				
Autumn	−1.00	120.00	0.097	0.496***
	(175.63)	(93.16)	(0.216)	(0.134)
Spring	−24.00	45.00	0.138	0.311**
	(158.70)	(88.96)	(0.196)	(0.128)
Summer	34.00	120.67	0.282	0.549***
	(153.75)	(84.78)	(0.190)	(0.122)
Constant	−172.13	−355.01***	−2.12	−7.86
	(230.86)	(136.36)	(3.31)	(2.35)
Observations	54	48	54	48
F-test			16.41	59.18
Pseudo *R*-squared	0.339	0.613		
R-squared			0.631	0.876

LAD = least absolute deviation regression; OLS = ordinary least squares regression.
Note: Standard errors in parentheses, ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$.

models are reported. Notably, in Column 4, we find evidence of a seasonal effect, with three-month rolling average of Pb in vacuum dust significantly higher in autumn, spring and summer as compared to the reference season of winter.

Figs. 3 and 4, present best-fit linear solutions of the association between soil Pb and exterior dust gauge Pb (Fig. 3), and soil Pb and vacuum dust Pb (Fig. 4) by season. These figures show that the soil Pb effect appears to amplify (slopes rise and steepen) in the summer period as compared to the winter period. Such curve behaviours by season are consistent with a soil dust re-suspension hypothesis.

Next, we analysed the quarterly variation in the Pb content of petri-dishes in sampled houses as a function of soil Pb and exterior dust gauge Pb. In Column 1 and 2 of Table 4, results from least absolute deviation regressions are reported. We find that unit increases in soil Pb ($b = 0.035$, $p < 0.1$) and exterior dust gauge Pb

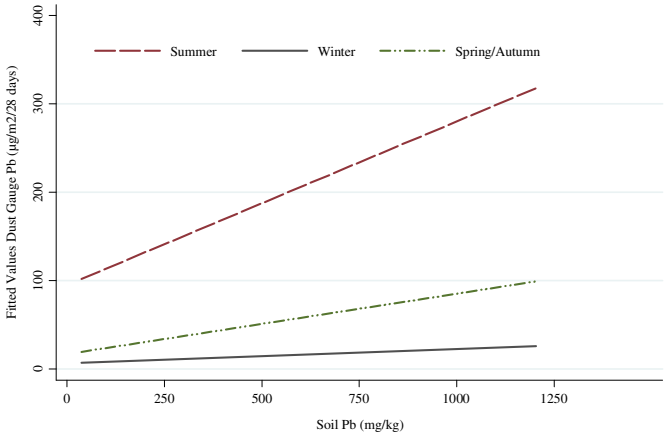

Fig. 3. Soil Pb concentration versus atmospheric Pb loading rates. This figure displays the relationship between soil Pb concentration and atmospheric Pb loading rates (µg/m^2/28 days) for the five houses during summer, winter and spring/autumn. Results indicate that the relationship is strongest during the summer.

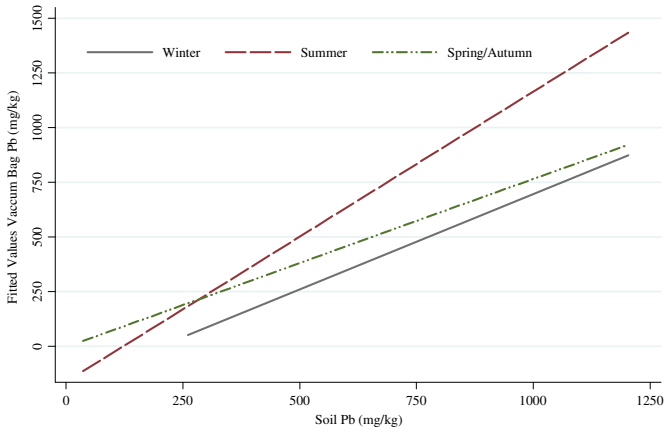

Fig. 4. Soil Pb concentrations versus vacuum Pb concentrations. This figure displays the relationship between soil Pb concentration (mg/kg) and vacuum Pb concentrations (mg/kg) for the five houses during summer, winter and spring/autumn. Results indicate that the relationship is strongest during the summer. The slope between summer is statistically different than the slope for winter and spring/autumn.

($b = 0.163$, $p < 0.05$) are associated with increases in the median of the quantity of Pb accumulated in petri-dishes. In Columns 3 and 4 of Table 4, least squares regression model results are shown. In Column 3, we find that a 1% increase in soil Pb increases the expected quantity of petri-dish Pb by 1.8% (where $p < 0.001$). The results show that a 1% increase in exterior dust gauge induces a more modest increase of 0.166% ($p < 0.05$) in petri-dish Pb (column 4, Table 4). With the exception of model 3, effects of seasonality on petri-dish Pb loadings are not significant.

3.2. X-ray Absorption Spectroscopy analysis of soil and vacuum dust

3.2.1. Soils

The two soil samples, from houses A and C, exhibit simple spectra characterized by a relatively featureless major "beat" pattern, as observed in Fig. 5. Photoelectron backscatter interference from the closest near-neighbour shell of oxygen atoms yields this simple beat pattern. The pattern indicates a lack of significant electron wave backscatter from second-, third-, or fourth-shell Pb or other heavy-element neighbours. This suggests that Pb is sorbed onto a light-element matrix, e.g., organic matter. In contrast, the repetitive physical structure (spatial arrangement of atoms) in crystalline Pb compounds yields complex spectra due to additional backscatter from Pb near-neighbour shells (see, for examples, the hydrocerussite and Pb oxide spectra). The higher frequency spikes in the samples at about 7k are artefacts due to crystal glitches.

The spectral patterns can be treated as "fingerprints", to be compared to spectra of modelled compounds (Pb humate, Pb particulate matter, hydrocerussite and Pb oxide) of known composition. The similarities of the Pb-humate and Pb-particulate matter are obvious as are the contrasts to hydrocerussite and PbO (Fig. 5). Fig. 5 illustrates a close match between the house C soil sample to Pb-humate and also the house A soil sample to Pb-particulate organic matter. These model compounds are similar: Pb sorbed onto humic acids and Pb sorbed onto organic (plant leaf) particles.

Spectral fits to other Pb species with similar simple beat spectra cannot be ruled out, given that the spectra extend only to 8k. Spectra similar to those of our soils can be produced by Pb sorbed on other forms of organic matter, or on clays that lack stronger backscattering metals, e.g., iron. However, we can say with a high degree of certainty, that the soil Pb is present in a sorbed form, which is most likely the result of reaction and alteration of the form of the Pb after it was introduced or deposited in the yards.

3.2.2. Vacuum dust

The spectra of the three vacuum dust samples, from houses A, C, and D, are similar in their main features, with some secondary differences (Fig. 5). Deviation from the simple beat pattern of the organic sorption model compounds is evident in house D dust in the narrowing or infilling of the 'valley' between the second and third spectral peaks, at about 4-5k. This indicates the presence of a strong backscattering element(s) beyond the first-neighbour shell. A mixture of 62% Pb sorbed on particulate organic matter and 38% hydrocerussite provides a close fit to house dust D (Fig. 6). These compounds were chosen from numerous combinations of up to three model compounds from in-house our spectral library of Pb compounds associated commonly with paint and urban pollution (analysed by the University of Texas at El Paso (UTEP) team at the Stanford Synchrotron Radiation Lightsource). Furthermore, hydrocerussite, or 'white Pb', was the dominant species of Pb in the commercial Pb-based paints of the 20th Century (See et al., 2007).

Fig. 6 indicates that the spectrum of house C vacuum dust closely matches that of Pb sorbed on particulate organic matter, and house A vacuum dust lies between the compositions of C and D (Fig. 6). Thus, the majority of the Pb in the household vacuum dusts has an origin that appears to be external to the home and has probably been either tracked in by foot traffic, or entered the homes as airborne soil and dust. The remainder appears to be old Pb-based paint, which probably originated predominantly inside the house given that the two associated soils did not contain Pb-based paint.

3.3. Pb isotope analysis of vacuum dust

Fig. 7 visually summarizes the Pb isotopic composition ($^{207}Pb/^{204}Pb$ versus $^{206}Pb/^{204}Pb$) data for interior vacuum dust, surface (0–2 cm) and subsurface soil (0.5 m), paint chips, and petrol Pb isotopic composition. To aid interpretation we have also included relevant data on paint and leaded petrol from relevant published sources (Gulson et al., 1983, 1995, 2003, 2006a,b, 2008). In Fig. 7 the vacuum Pb isotope composition for houses A, C, and D

Table 4
Least squares and least absolute deviation coefficients predicting Pb content of petri-dish as a function of soil Pb and exterior dust gauge Pb. This table presents the statistical results of least squares and least absolute deviation regressions predicting the Pb content of Petri-Dish as a function of soil Pb and exterior dust gauge Pb.

	LAD petri-dish	LAD petri-dish	OLS *ln* petri-dish	OLS *ln* petri-dish
Soil Pb	0.035*			
	(0.020)			
ln soil Pb			1.801***	
			(0.500)	
Dust gauge Pb		0.163**		
		(0.08)		
ln dust gauge Pb				0.166**
				(0.07)
Interior (Reference)				
Attic	18.32	1.17	2.242**	2.25*
	(15.60)	(12.55)	(1.084)	(1.17)
Quarters 2, 3, 4 (Reference)				
Quarters 1, 5	14.67	−2.61	2.567**	1.17
	(39.51)	(16.38)	(1.126)	(1.39)
Constant	−15.95	0.00	−13.21***	−1.78*
	(16.82)	(10.83)	(3.095)	(0.99)
Observations	44	44	44	44
F-test			7.91	4.83
Pseudo *R*-squared	0.120	0.160		
R-squared			0.160	0.266

LAD = least absolute deviation regression; OLS = ordinary least squares regression.
Note: Standard errors in parentheses, ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$.

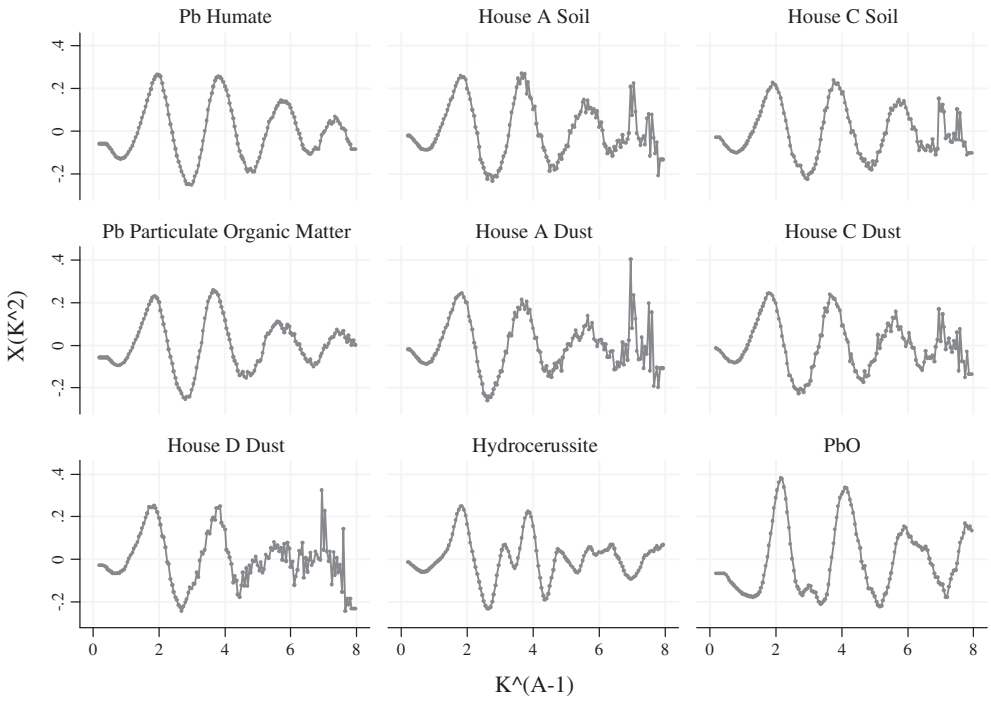

Fig. 5. XAS Pb speciation chart. This figure displays the Pb Spectra for houses A and B soil samples, houses A, C and D vacuum dust samples, Pb humate and Pb particulate organic matter, hydrocerrusite and PbO (lead oxide). The Pb spectra for Pb Humate, House A soil, House B soil, Pb particulate organic matter, House A dust and House C dust are very similar, while being dissimilar to typical paint Pb compounds – hydrocerussite and PbO. The spectra for House D is somewhat similar to the Pb spectras for House A dust and House C dust, while also being dissimilar to typical paint Pb compounds – hydrocerussite and PbO.

correspond more closely to the soil Pb isotope composition values than house-specific paint Pb isotope composition values. To estimate isotope ratio similarity, we calculated the Euclidean distance of each vacuum Pb isotope ratio to corresponding paint, surface soil, soil (0.5 m), and petrol isotope ratio values. Table S1 reports the Euclidean distance between the points where Pb isotopes plot on the Pb isotopes charts. In each house where Pb isotopes were analysed we find that vacuum Pb isotope composition ratios are more similar to surface soils than to subsurface soil (0.5 m) and paint chip samples (Fig. 7).

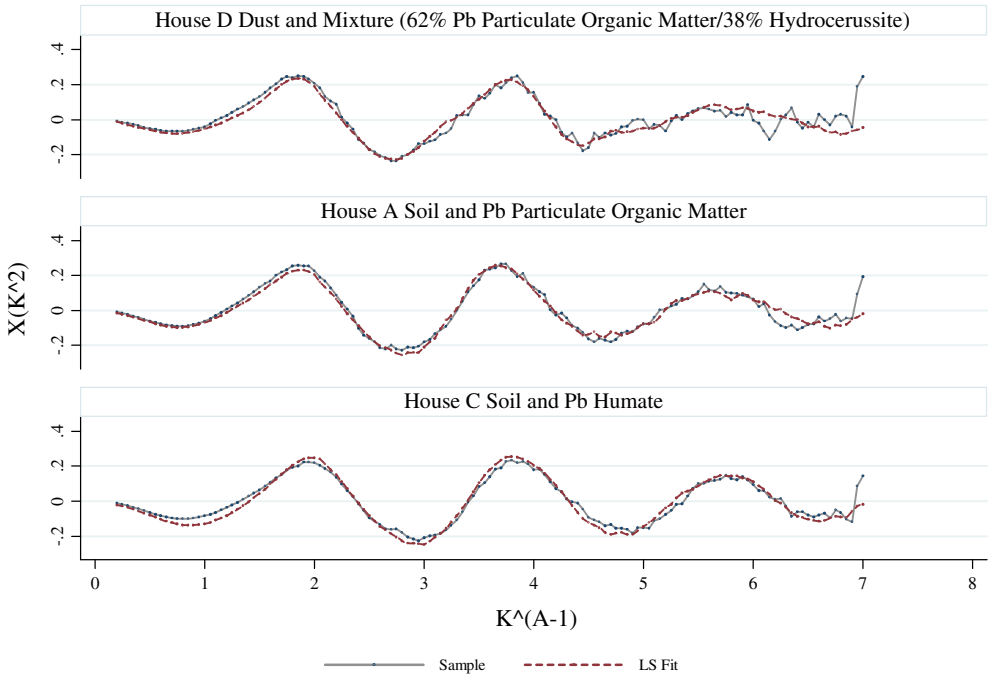

Fig. 6. XAS Pb speciation chart – paired samples. The chart displays the Pb Spectra for House D and the best fit line for Pb particulate organic matter (62%) and hydrocerussite (38%). The chart also displays a good fit between Pb spectra for house A soil and particulate organic matter. The chart also displays a good fit between the Pb spectra for house C soil and Pb humate.

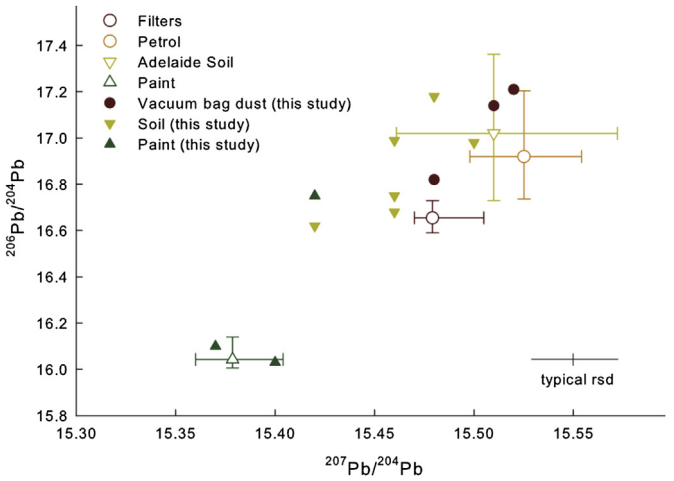

Fig. 7. Scatterplot of $^{207}Pb/^{204}Pb$ by $^{206}Pb/^{204}Pb$. This chart plots the $^{207}Pb/^{204}Pb$ ratio and the $^{206}Pb/^{204}Pb$ ratio of vacuum dust samples, surface soil sample, soil (0.5 m depth) samples and paint samples from houses A, C and D. In addition, Pb isotope ratios from paint samples collected from one house near Sydney (Gulson et al., 2003) and historical petrol Pb samples Pb isotope ratios (Gulson et al., 1983, 1995, 2006a,b) from the Sydney area are plotted as well. The results indicate that the Pb paint samples cluster together in the lower left part of the chart and the Pb isotope ratios for the Sydney petrol and the vacuum dust samples, surface soil sample, soil (0.5 m depth) samples from houses A, C and D cluster to the upper right portion of the chart. This suggests that the origin of the Pb in the houses is from petrol sources.

3.4. Lead deposition rates in house petri-dish, house attic and exterior dust gauge

Fig. 2 details the exterior dust gauge Pb deposition rates in houses A through E between November 2010 and January 2012 (see also Table S3 for additional information). Table 5 displays average quarterly Pb deposition rates for house petri-dishes (HS), attic petri-dishes (AT) and exterior dust gauges (DG) in houses A through E. In general, Pb deposition rates follow the following pattern: exterior dust gauge > attic > house. Interior and exterior Pb loading rates are correlated with exterior soil Pb concentrations and temporal Pb loading rates are highest in the summer and autumn and lowest in the winter and spring (Table 5, Fig. 3), which is consistent with a soil dust re-suspension hypothesis during the drier summer period (Laidlaw et al., 2005, 2012; Laidlaw and Filippelli, 2008; Zahran et al., 2013).

Percentiles for house dust concentration, exterior Pb loading rates, interior petri-dish Pb loading rates and attic petri-dish Pb loading rates are presented in supplementary Table S4.

4. Discussion

Soil Pb concentrations, exterior atmospheric Pb loading rates (dust gauge), interior vacuum dust Pb concentrations and interior petri-dish Pb loading rates correlate ($p < 0.05$) (Tables 2–4). This suggests that re-suspension of exterior soil is a primary source of Pb in the urban homes studied here. Other studies have shown that Australian soil Pb and children's PbB are correlated (Fett et al., 1992; Galvin et al., 1993; Willmore et al., 2006), but the specific pathway of re-suspension and airborne migration into urban homes was not identified, unlike in the USA (Hunt et al., 2006; Layton and Beamer, 2009; Hunt and Johnson, 2012).

In order to evaluate whether interior household Pb is linked to outdoor soil Pb we have examined multiple lines of evidence and have determined the following:

The correlation between exterior soil and interior vacuum dust concentration in this study ($r = 0.659$, $p < 0.001$) was also observed

Table 5
Pb deposition rates – external dust gauge, attic petri-dish and house interior petri-dish. This table presents quarterly average Pb deposition rates collected for the external dust gauge, attic petri-dish and interior petri-dish's collected at 5 houses between November 2010 and January 2012.

Sample location	Season (Quarter)	House A	House B	House C	House D	House E
Interior	Summer1	24	<2	38	<2	NA
Interior	Autumn	22	<2	27	15	NA
Interior	Winter	<2	<2	52	<2	<2
Interior	Spring	<2	<2	<2	<2	<2
Interior	Summer2	NA	NA	14	38	<2
Attic	Summer1	103	<2	917	98	NA
Attic	Autumn	17	12	91	43	NA
Attic	Winter	72	<2	<2	<2	<2
Attic	Spring	<2	<2	123	41	<2
Attic	Summer2	30	<2	244	156	30
Exterior dust gauge	Summer1	354	74	155	265	NA
Exterior dust gauge	Autumn	97	51	162	120	NA
Exterior dust gauge	Winter	<2	<2	12	<2	NA
Exterior dust gauge	Spring	90	85	84	<2	<2
Exterior dust gauge	Summer2	195	38	246	139	16

Units = micrograms/m^2/28 days; Quarterly Dust Gauge Samples = average of three monthly dust gauge samples; Quarterly Interior and Attic Samples = 1 quarterly sample each.

in other locations by Bornschein et al. (1986) ($r = 0.75$) and Thornton (1990) ($r = 0.531$, $p = 0.001$, $n = 4512$). The correlation between exterior atmospheric Pb loading and interior vacuum Pb concentration in this study is significantly positive ($r = 0.314$, $p < 0.001$). Adjusting for season of observation, least absolute deviation regression models find that per unit of increase in soil Pb loading increases the median Pb content of vacuum dust by 0.369–1.238 mg/kg. The temporal flux of interior vacuum dust Pb concentrations and exterior dust gauge loadings indicates strongly that exterior soil re-suspension is the driver of the seasonal variations (Figs. 3 and 5). Further, soil Pb concentrations are significantly correlated with the natural log of interior petri-dish Pb loading rates ($p < 0.001$; see Table 4).

Interior vacuum Pb isotope ratios plot closer to the exterior surface soils than interior paint Pb isotope ratios indicating that the source of interior Pb is from exterior surface soils (Fig. 4). In addition, XAS spectra of the exterior surface soil and the interior vacuum dust were nearly identical in the most contaminated houses (Houses A and C) – indicating that they originate from the same source. In House D the data show that the source of the Pb in the house dust was from Pb in soil and house paint (Fig. 3). Finally, in terms of examining the possibility that paint was a primary source of household Pb, the study data show that vacuum dust Pb concentration and paint Pb are not correlated, except when a 3 month moving average is applied (Table 3).

The implications from this study are that in old, densely populated urban areas like Sydney, elevated soil Pb appears to be migrating into homes. The transfer of contaminants from external to internal environments is associated with elevated interior dust Pb concentrations, which in turn have significant potential to raise PbB levels (Layton and Beamer, 2009). If interior dust Pb concentrations similar to the levels found at higher concentrations in this study are widespread in Sydney (and the prevailing soil Pb research indicates that this is the case for the inner west of Sydney – population 2.02 million; NSW Government, 2013), there is a potential for widespread, low-level Pb poisoning, especially in areas with a history of high traffic rates (cf. Laidlaw and Taylor, 2011).

The risk at the lower end of the exposure spectrum is significant because the slope between soil Pb and PbB is steep at low concentrations, resulting in a rapid uptake of Pb in blood as soil Pb levels increase (Mielke et al., 2007). Importantly, the greatest relative decline of IQ due to Pb exposure occurs at the lowest Pb

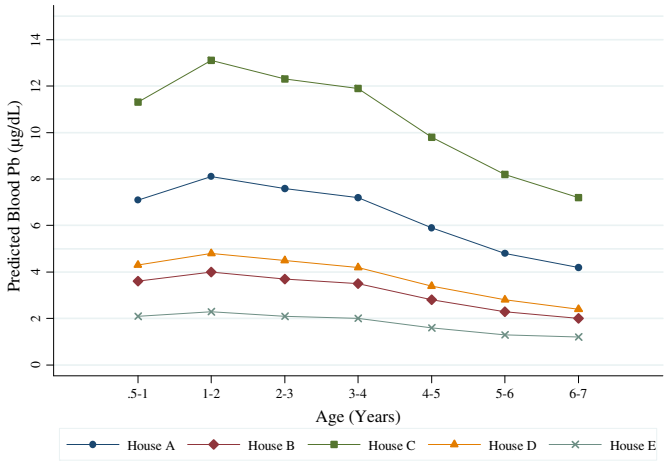

Fig. 8. IEUBK modelled PbB. Plot of predicted childrens's (age 0–7) PbB in study homes A through E which was calculated using the Integrated Exposure Uptake Biokinetic (IEUBK) model (see text for input values).

exposures (Canfield et al., 2003; Rothenberg and Rothenberg, 2005). In terms of population exposures, Taylor et al. (2012) estimated that approximately 100,000 Australian children aged 0–4 years may have PbB levels associated with adverse health outcomes, but because there is no routine or systematic PbB testing of city children in Australia the potential risk of exposure is unconstrained. We estimate exposures in children aged 0–7 in houses A through E using our data via the IEUBK model (USEPA, 2013a) (Fig. 8). This model predicts that children aged 0–7 years in houses A and C have the potential to accrue a PbB >5 µg/dL. Thus, the data from this study supports the argument that both soil lead and blood lead should be evaluated in a systematic manner across our older inner cities to decipher accurately the real risks to young children (Laidlaw and Taylor, 2011). This study supports primary prevention, and precedence for soil intervention as a method of primary prevention has been established by the Norwegian Government whereby soil Pb intervention is undertaken without measuring Pb exposure of children (Ottesen et al., 2008).

4.1. A possible way forward for dealing with potential futures exposures

In New South Wales (NSW), section 149 Planning Certificates are issued in accordance with the *Environmental Planning & Assessment Act 1979* (NSW). The certificates contain information on how a property may be used and the restrictions on development. When land is bought or sold the *Conveyancing Act 1919* (NSW) requires that a Section 149 Planning Certificate be attached to the Contract for Sale. Under sections 149 (5) and (6) of the *Environmental Planning & Assessment Act 1979* (NSW) there is a clear opportunity to identify actual or potential contamination at properties:

(5) A council may, in a planning certificate, include advice on such other relevant matters affecting the land of which it may be aware.
(6) A council shall not incur any liability in respect of any advice provided in good faith pursuant to subsection (5). However, this subsection does not apply to advice provided in relation to contaminated land (including the likelihood of land being contaminated land) or to the nature or extent of contamination of land within the meaning of Part 7A.

Therefore, we contend that following proper environmental assessment of potentially contaminated areas and properties for dust or soil lead issues, a section 149 certificate should include the assessment information so that prospective purchasers are aware of any potential risk to health.

4.2. Study limitations

There are some contradictions within the data. The atmospheric Pb loading pattern clearly demonstrates that Pb loading rates are highest in the summer and autumn and lowest in the winter. While this is in agreement with temporal Pb concentration patterns identified in the United States (Zahran et al., 2013), it is the opposite signature to that identified in other air and dust lead seasonal studies in Sydney (Chiaradia et al., 1997; Cohen et al., 2005; ANSTO 2013). The ANSTO data reveals that the air Pb (µg/m^3) peaks in May and June, the opposite of the Pb loading rates (µg/m^2/28 days) observed in this study. Chiaradia et al. (1997) suggested that the air Pb concentration peak in the autumn-winter months are probably related to thermal inversion that is typical of Sydney's cooler winter season. Another contradiction is that the quarterly interior petridish Pb loading rates did not display a summertime peak that was observed in the monthly exterior Pb loading rates (µg/m^2/28 days) and interior vacuum Pb concentrations (mg/kg). These contradictions further support the need for expanding the study to more Sydney homes to confirm the trends identified within the small group of homes in the study.

5. Conclusion

This study used multiple types of environmental analysis to show the main source of Pb found within interior dust at four inner-city Sydney homes is derived from Pb that has been tracked-in and re-suspended from exterior soil. Lead paint was shown to be a partial source in one of the three homes where Pb isotopes were sampled and Pb speciation was analysed. This study confirms that exterior soil Pb is being transferred to domestic interiors. Given that the greatest relative uptake of Pb into blood occurs at the lowest soil Pb concentrations and the greatest relative decline of IQ due to Pb exposure occurs at the lowest Pb exposures, then Pb contaminated urban roadside and garden soils may require remediation or isolation to provide a margin of safety for children.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at doi:10.1016/j.envpol.2013.09.003.

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Chapter 2 - Paper 3: "*Response to Brian Gulson's - Comments on: Identification of lead sources in residential environments: Sydney Australia."*

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Reply to letter to the editor

Reply to comments on "Identification of lead sources in residential environments: Sydney Australia" by Laidlaw et al. (2014)

We would like to start by thanking Brian Gulson for taking time to highlight his concerns and bringing our findings to the attention of *Environmental Pollution* readers.

In constructing our response to Brian Gulson's commentary, we had difficulty in deciphering which of Gulson's (2014) "*submitted for publication*" references were being referred to. Gulson referred an article submitted for publication in three ways, making it difficult to ascertain which one to respond to: 1) (Gulson et al., submitted for publication), 2) (Gulson et al., 2006, submitted for publication), and 3) (Gulson et al., 2013). Therefore, we have had to use our best guess as to which publication(s) that we were intended to respond to.

In the following text, Professor Gulson's comments have been placed in italics and in a bold font.

The terminology "Pb poisoning" is alarmist especially for parents of young children and has been discontinued by, for example, US Centers for Disease Control and Prevention in their current recommendations at low blood Pb (PbB) levels and by respected experts in the Pb field (e.g. Canfield et al., 2003).

While we prefer to use the term "lead poisoning" in our paper, we acknowledge that there is no international consensus on its precise definition. However, we defend its use because it brings to more immediate attention to the well-known risks associated with lead exposure, particularly in children at very low levels (i.e. <10 μg/dL) (see for example United States National Toxicology Program, 2012). The dangers of exposure to environmental lead (Pb) have been down-played consistently in the Australian context (Gulson et al., 2008; Taylor and Schniering, 2010; Taylor et al., 2011, 2012b, 2013; Taylor, 2012; Mackay et al., 2013), which has not been overly helpful in addressing legacy Pb issues in urban cities or locations associated with metal mining and smelting activities. Similar challenges have been identified in the United States context where lead paint exposures have been particularly problematic (Bellinger and Bellinger, 2006; Markowitz and Rosner, 2013).

The Australian National Health and Medical Research Council (2009) does not refer to Pb poisoning directly but refers to acute effects at levels of 70–100 μg/dL and longer-term effects from much lower levels of exposure i.e. at 10 μg/dL or lower. Bellinger and Bellinger (2006) summarize the spectrum of Pb exposure effects and identify that developmental toxicity including decreased IQ, hearing, growth, impaired peripheral nerve function and transplacental transfer is evident at exposures below 10 μg/dL of PbB (see also United States National Toxicology Program, 2012). The recent WHO Childhood Lead Poisoning report (2010, p. 12) makes the following comments with respect to the adverse effects of Pb exposure: "*Recent research indicates that lead is associated with neurobehavioural damage at blood levels of 5 μg/dl and even lower. There appears to be no threshold level below which lead causes no injury to the developing human brain.*" We note that other world experts such as Professor Bruce Lanphear prefer to use the phrase "lead toxicity" (e.g. Lanphear et al., 2005, 2008). While the term 'toxicity' avoids any distracting and unhelpful arguments about terminology, its usage is synonymous with 'poison'.

Notwithstanding these comments over the use of nouns with lead, we contend that our use of the phrase 'childhood lead (Pb) poisoning' is appropriate and is also consistent with the ordinary English meaning and definition of the word poison: "*a substance that when introduced into or absorbed by a living organism causes death or injury*" (Allen, 1990, p. 920). The use of the word poison is accurate and unambiguous and prevents one from erring towards a situation that could be interpreted as down-playing the risk of lead exposure at any level.

Finally, in terms of the most recent CDC commentary, perusal of their website (CDC, 2014) covering Pb shows that under the title "*Childhood Lead Poisoning*", they state the following: "*Approximately 500,000 U.S. children aged 1-5 years with blood lead levels above 5 micrograms of lead per deciliter of blood, the reference level at which CDC recommends public health actions be initiated. Lead poisoning can affect nearly every system in the body. Because lead poisoning often occurs with no obvious symptoms, it frequently goes unrecognized. Lead poisoning can cause learning disabilities, behavioral problems, and, at very high levels, seizures, coma, and even death.*"

The results of the first 18 months of our study (Gulson et al., 2006) showed a geometric mean PbB value of 2.6 μg/dL and, for the 5 year time frame, the geometric mean was 2.1 μg/dL the same (Gulson et al., submitted for publication). These values are 1/3 of the current recommendations of the Australian National Health and Medical Research Council and half the current reference value of 5 μg/dL of the US Centers for Disease Control and Prevention.

The Gulson et al. (2006, p. 110) paper states the following: "*Six of the 114 children (5%) had a PbB >10 μg/dL, the current Australian National Health and Medical Research Council guideline. Three of these children had a PbB >15 μg/dL, requiring notification to the NSW Health Department.* While we acknowledge that the Gulson et al. (2006) paper is excellent, a sample size of 114 children is likely to be too small to be numerically and spatially representative of the PbB average 813,200 children that live in Sydney (ABS, 2014). Indeed, similar criticisms of a slightly larger blood lead study of Sydney's children ($n = 158$; Fett et al., 1992) of which Gulson was a co-author, were made in 1993 (Aldrich et al., 1993). The Gulson

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et al. (2008) study used a sample size of between approximately 15 and 95 children at various six-month sampling intervals where 665 PbB measurements recorded a PbB mean of 2.57 μg/dL (total cohort over the study period consisted of a maximum recruit of 51 females and 56 males). Of these 665 samples, all the PbB samples that exceeded 10 μg/dL ($n = 8$) were omitted (Table 2, p. 209). In the absence of a recent and comprehensive PbB survey in Sydney i.e. a study that includes more children, we suggest that the use of the study's mean blood level (2.57 μg/dL) may not represent accurately the central tendency of PbB level for Sydney children. While not intending to detract from the quality of the study itself, the reality is that the Gulson et al. (2008) study is an ad-hoc sample and cannot be construed as being representative of the mean PbB of Sydney's children. It is worth noting that of the 107 children sampled in the Gulson et al. (2008) study, 7.5% (8 children) returned a blood lead level >10 μg/dL. This proportion of children is consistent with the conclusion drawn about contemporary elevated PbB exposures in Australian children by Taylor et al. (2012a). This article estimated that around 100,000 Australian children may have PbB levels >5 μg/dL, which is the reference level used by the USA's CDC to initiate public health actions.

By comparison, in New Orleans (USA) children's geometric average PbB of 5.68 μg/dL was determined using a database of 55,551 children (Zahran et al., 2013b) distributed over 286 census tracts. New Orleans has similar soil Pb concentrations to Sydney (Birch et al., 2011; Zahran et al., 2013b). New Orleans children currently have a PbB prevalence (>5 μg/dL) of 29.6% (Mielke et al., 2013). In Detroit, 33% of 367,839 children had PbB >5 μg/dL (Zahran et al., 2013a), however Detroit has soil Pb concentrations lower than Sydney (Murray et al., 2004). The reason for the large datasets in the USA is that PbB is measured by state governments while in Australia governments do not measure routinely PbB levels in all children (Laidlaw and Taylor, 2011).

Evaluation of the full 5-year results shows that the predictor variables of diet, interior house dust loadings, handwipes, exterior dust sweepings and soil can only explain 9% of the variance in PbB (Gulson et al., submitted for publication). This low value contrasts with the very high contribution (40%) of soil to PbB found in a New Orleans study (Zahran et al., 2013b) for soils of similar Pb concentrations.

We could not locate any "5 year results" published in Gulson's journal articles (Gulson et al., 2006, 2008) that measured a 9% variance in PbB due to predictor variables of diet, interior house dust loadings, hand-wipes and exterior dust sweepings. Therefore we are unable to comment. Gulson et al. (2013) notes that the contribution of dust lead to PbB is 59%.

Gulson's (2014) stated figure of 40% for the variance in the Zahran et al. (2013b) study is *incorrect*. In the Zahran et al. (2013b) paper, soil Pb concentration explained 75.4% of the between neighborhood variation in PbB levels in a dataset of 55,551 children in metropolitan New Orleans, USA. The dataset in this study included 55,551 children and 5467 surface soil samples (19 surface soil samples per census tract). The 40% figure refers to the combined explained variance of residential and busy street soil samples, derived from a variance partitioning procedure.

Turning to the data for the environmental samples, Laidlaw et al. (2014) provide new Pb isotopic ratios, measured by the medium quality ICP-MS method, for 3 samples of vacuum cleaner dust, 3 samples of paint and 6 soil samples. The authors concluded that there was a similarity in Pb isotopic ratios of the vacuum cleaner dust and soils and the source was from gasoline rather than from paint. Two samples of paint had low $^{206}Pb/^{204}Pb$ ratios of approximately 16.7. Unfortunately they ignored the detailed study of 3 houses in the inner Sydney suburbs which specifically focused on microscopic identification of individual paint layers and their Pb isotopic compositions (Gulson et al., 1995). In their study up to 13 layers of Pb paint were identified from 1 house and had $^{206}Pb/^{204}Pb$ ratios ranging from 16.0 to 18.5. A mixture of such layers in a bulk paint would result in $^{206}Pb/^{204}Pb$ ratios approximating those shown in Figure 3 (plotted in an unusual axis notation compared with conventional Pb isotopic studies) of Laidlaw et al. for soil, gasoline and paint.

Gulson et al.'s (1995) Pb isotopic data only contained Pb paint with an isotopic ratio that was plotted on a single axis – $^{206}Pb/^{204}Pb$. The bulk of the Pb source isotopic composition data (filters, petrol, soil and paint) we used in the Laidlaw et al. (2014) were plotted with the benefit of using Pb isotopic ratios on *two* axes – $^{207}Pb/^{204}Pb$ and $^{206}Pb/^{204}Pb$. The Pb paint isotopic source data that originated from the Gulson et al. (2004) paper that was used to compare to the Pb paint isotopic composition ratios from the Laidlaw et al. (2014) study were presented on *two* axes – $^{207}Pb/^{204}Pb$ and $^{206}Pb/^{204}Pb$. Furthermore, two of our Laidlaw et al. (2014) Pb paint samples plotted within the 95% confidence interval of Gulson et al.'s (2004) house Pb paint isotope ratios. Regarding Gulson's (2014) comment that we used "*unusual axis notation compared with conventional isotopic studies*", it is worth noting that we used the same isotopic ratios as used in Gulson et al. (2004) but with the values placed on the opposite *x* and *y* axes.

Such samples [environmental samples] ***may contain Pb paint both from renovations of the studied houses or from neighboring houses undergoing renovations or just deterioration of Pb paint as shown by Gulson et al. (1995) and in the US (e.g. Farfel et al., 2005).***

None of the adjacent brick or masonry homes underwent renovations during the study. Further, none of the neighbouring properties were constructed of painted brick. In addition, none of the homes examined underwent renovations during the study with the exception of House D that had some internal drywall replaced in a portion of the home. At this house Laidlaw et al. (2014) concluded that the Pb in the house dust originated from both Pb in soil and Pb paint sources.

Laidlaw et al. concluded that the source of interior dust Pb was primarily from soil in 2 of 3 houses. In our 5-year study we found that interior dust fall accumulation was significantly related to exterior dust sweepings (p 0.003) but not soil (p 0.165) (Gulson et al., submitted for publication).

Gulson appears to have made a mistake when referring to his "5-year study" as Gulson et al. submitted for publication, because the results for this study were published in Gulson et al. (2006) and Gulson et al. (2008). We note that the Gulson et al. (2006) 5-year study observed that Pb concentration in soil was the only significant predictor ($p \leq 0.001$) for Pb in dustfall accumulation inside the Sydney study homes (Gulson et al., 2006) (Table 3, p. 106). Further, Gulson et al. (2006) states that dustfall Pb accumulation was a significant predictor for Pb in interior hand wipes ($p = 0.014$) for children prior to playing outside (Table 106, page 107, Gulson et al. (2006). Indeed, the Abstract of Gulson et al. (2006) states that "*The only significant predictor for PbB was dustfall accumulation…*". Therefore, based on Gulson's own data (Gulson et al., 2006), Pb in interior dustfall is associated with soil Pb concentrations, and Pb dustfall accumulation is a significant predictor for children's PbB. Consequently, in light of this, it is not clear what specific point Gulson is trying to make about our observations given that we found that soil Pb is the source of Pb in interior house dust in two out of three brick Sydney homes in the Laidlaw et al. (2014).

Using X-Ray Absorption Spectroscopy with a Synchroton [sic] Laidlaw et al. identified the species of Pb in vacuum cleaner dust and surface soils from 3 houses. As this is a relatively new and exciting approach and not widely available, it might have

been appropriate that the authors compared their results with similar but more detailed studies undertaken by the Ottawa group and published in Environmental Science and Technology (Rasmussen et al., 2011; MacLean et al., 2011; Walker et al., 2011).

These studies are excellent. We note that our paper was not a review of the literature on the topic but that these are useful suggestions.

Using an excellent monthly collection sampling protocol Laidlaw et al. found that the average Pb levels in vacuum cleaner dust were significantly higher in autumn, spring and summer compared with winter. Even though they observed a strong association of Pb in soil and interior petri dust loadings and a moderate association for exterior dust gauge loadings and interior dust [Pb concentrations] there was no significant association of interior dust [Pb concentrations] with seasonality which is rather surprising give the vacuum cleaner dust results.

In order to assess the interior dust Pb seasonality and any correlation between the interior dust Pb concentrations and interior petri dish Pb loading rates (airborne Pb), other monitoring methods may be required that are sensitive enough to identify any extant seasonality signal in the interior atmospheric dust fraction. Such equipment might include volumetric air monitoring devices that measure airborne particulate Pb mass per unit volume per unit of time or micro-orifice uniform deposit impactor (MOUDI) air samplers that permit the collection of multiple, but very precise size fractions of aerosols at multiple particle size cut-off points.

The authors had no explanation for why their temporal Pb concentration patterns were the reverse of previous Sydney studies.

In the Laidlaw et al. (2014) study, atmospheric Pb loading rates (mass/area/time) were measured. The "*previous Sydney studies*" (Chiaradia et al., 1997; Cohen et al., 2005; ANSTO, 2013) measured airborne concentration (mass/volume). Consequently, although the measurements cannot be compared directly Brian Gulson raises a useful point. We did grapple with this difference at the time of writing and concluded that there was no immediately obvious or useful answer, but nevertheless, we acknowledge that the issue warrants further research.

General comment

With regard to the Gulson et al. (2013) re-analysis of old (1994–1995) Australian data (Donovan, 1996) as referred to in Gulson (2014), we note that at the time of the data collection in the mid 1990s, Pb was still being used in motor vehicle gasoline (its use in Australian motor car fuel ceased in 2002). Consequently, this would render source apportionment conclusions from this data biased strongly towards gasoline emissions. This situation is very different from the present, where there is no added Pb used in motor vehicle gasoline and the only other similar current Pb source that is used is Pb added to general aviation gasoline (avgas) (USEPA, 2010), which is consumed in much smaller quantities.

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CHAPTER 3

Estimation of historical vehicle traffic Pb emissions in US and California urbanized areas and their legacy in urban soils and continued effect on children's health.

Chapter 3 estimates the mass of petrol derived Pb emitted historically into the atmosphere and soils in 90 US urban areas and major California cities (Mielke et al., 2010; Mielke et al., 2011). Historical soil Pb studies in the US and California are also presented and discussed (Mielke et al., 2010; Mielke et al., 2011).

Paper 1: "*Estimation of leaded (Pb) gasoline's continuing material and health impacts on 90 US urbanized areas."*

HW Mielke, **MAS Laidlaw**, CR Gonzales

Published in *Environment International* (2011).

This publication was undertaken because nobody had previously estimated the large volume of lead that had been emitted into US urban areas due to the use of leaded gasoline. This study also documented all the journal articles about diffuse urban soil lead contamination that were available in these cities. This work is important because it documents how urban soil lead in the US has been contaminated by the past use of leaded gasoline, not Pb paint.

Author Contributions (Mielke H.W., Laidlaw M.A.S., et al., 2011):

HW Mielke: 55%

Concept, lead emission estimate calculations, writing, editing.

MAS Laidlaw: 40%

Writing, literature review, editing.

CR Gonzales: 5%

Figures

Paper 2: *"Lead (Pb) legacy from vehicle traffic in eight California urbanized areas: continuing influence of Pb dust on children's health."*

HW Mielke, **MAS Laidlaw**, CR Gonzales

Published in *Science of the Total Environment* (2010).

This paper estimates the quantity of Pb emitted into the atmosphere and soils in California's largest cities. It also reviews the literature about soil Pb and PbB studies in California. It is important because it demonstrates that soils in urban California areas have been contaminated from past use of Pb in gasoline which continues to pose a human health hazard to children in California.

Author Contributions (Mielke H.W., Laidlaw M.A.S., et al., 2010):

HW Mielke: 55%

Concept, lead emission estimate calculations, writing, editing.

MAS Laidlaw: 40%

Writing, literature review, editing.

CR Gonzales: 5%

Figures.

Chapter 3 - Paper 1: "*Estimation of leaded (Pb) gasoline's continuing material and health impacts on 90 US urbanized areas."*

Authors: HW Mielke, **MAS Laidlaw**, CR Gonzales

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Review

Lead (Pb) legacy from vehicle traffic in eight California urbanized areas: Continuing influence of lead dust on children's health

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ABSTRACT

This article describes the magnitude of U.S. lead (Pb) additives in gasoline from 1927 to 1994 and estimated quantities of Pb dispersed by vehicle traffic in eight urbanized areas (UAs) of California from 1950 to 1982. The findings are the basis for predicting the health impact of Pb on children living in UA of California. Quantitative U.S. national data for 1927–1994 were from the U.S. Senate hearing of the 1984 Airborne Lead Reduction Act. Vehicle traffic data, fuel efficiency, percentage leaded gasoline, and quantities of Pb in gasoline were obtained for 1982 from public and corporate records to estimate vehicle Pb emissions for small to very large UAs of California. California fuel consumption records and yearly quantities of Pb additives per gallon were the basis for estimating the 1950–1982 dispersion of Pb in each UA. Lead additives were calculated by multiplying annual vehicle fuel used by average Pb per gallon. The proportion of Pb additive for each UA was calculated from vehicle miles traveled (VMT) driven in 1982 divided by miles per gallon fuel consumption times the ratio of leaded to unleaded fuel times Pb additive per gallon. U.S. Environmental Protection Agency calculations of the fates of Pb were used to estimate Pb aerosol dispersal in each UA. About 108 billion miles of travel in 1982 within 8 UAs accounts for 3200 metric tons of Pb additives or ~60% total Pb additives in California. Between the 1950–1982 peak of Pb additives, about 258,000 metric tons are accounted for out of the state 412,000 metric tons total during the same time period. The estimates of the quantities of Pb dust that accumulated within various UAs in California assists with predicting the continuing influences of Pb on children's exposure. Mapping the soil Pb reservoir assists with establishing the priority for enhancing environments of children.

Contents

Abbreviations: ALRA, Airborne Lead Reduction Act; DVMT, Daily vehicle miles of travel; Miles per gallon, mpg. Fuel economy is defined as miles per gallon up gasoline; NHANES, National Health and Nutrition Examination Survey; UA, Urbanized area; U.S. gallon, 3.79 l; U.S. miles, 1.61 km.

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1. Introduction

The quantities of lead (Pb) additives and their dispersion within urbanized areas (UAs) are the topic of this article. The study site is California. Whether it is from deterioration or mismanagement of the large tonnages of intact Pb-based paint in residences, or the equally large tonnages of Pb dust from additives to gasoline, the ultimate results are the continuing exposure of children to Pb dust (Mielke and Reagan, 1998). Exterior soils represent an enormous reservoir of Pb, and indications of the size of the reservoir are noted by studies of children's hands before and after outdoor play and also by direct measurement of Pb loading on the soil surface (Viverette et al., 1996; Mielke et al., 2007b; Nielsen and Kristiansen, 2005). Exterior soils via track-in and resuspended Pb aerosols contribute substantially to the mass transfer of Pb dust into residential interiors (Clark et al. 2004; Caravanos et al., 2005; Hunt et al., 2006; Layton and Beamer, 2009).

At the urban scale, the extent of the impact of Pb additives in gasoline was indicated by findings in garden soils of Baltimore, Maryland (Mielke et al., 1983). Based on vehicle miles of travel, Pb additives in gasoline were calculated at 5000 to 10,000 metric tons for Baltimore City during the era of leaded gasoline (Mielke et al., 1983). The study concluded that the use of leaded gasoline produced a highly polluted inner city compared with a less polluted outlying community and may be an important factor in the childhood Pb exposure problem (Mielke et al., 1983). In the South Coast Air Basin of California the quantities of Pb additives used in gasoline from 1970 to its ban were estimated at 20,000 metric tons (Harris and Davidson, 2005). Because highways were commonly planned and constructed to move traffic into and through cities, UAs of California probably have the same soil Pb and Pb exposure characteristics as described for other UAs (Post, 2007).

The objectives of this article are: 1) to provide an overview of the quantities of Pb additives used in vehicle fuel in the U.S. from 1927 through 1994; 2) to evaluate Pb additives dispersed in 1982 by vehicle traffic in 8 UAs of California; 3) to estimate the total Pb dispersed in each of the 8 UAs from 1950 through 1982, the period bracketing the peak use of Pb additives, and 4), to provide a review of soil Pb and predict the impact of the accumulated Pb on children's Pb exposure for each UA. Fulfilling these objectives should provide characteristics about Pb additives as a function of UAs and residential community location and result in better understanding of the remediation needs to protect children from exposure to Pb dust.

2. Methods

Three datasets were developed. First, the U.S. nationwide quantities of Pb additives in gasoline were obtained from corporate and government reports. Data were available from 1927 through 1994, when gasoline contained Pb additives. Second, the 1982 quantities of Pb additives used in eight UAs were calculated from data available from a corporate report and publicly available records for eight cities of California (Ethyl, 1982; U.S. DOTa, 1987). Third, the 1982 proportions of Pb were applied to California data on fuel consumption from 1950 to 1982 to calculate the approximate quantities of Pb used in each of the same 8 California UAs.

2.1. Annual quantities of lead additives in gasoline, 1927–1994

Data of the annual amounts of Pb from 1927 to 1984 were obtained from a figure included in testimony by Ethyl Corporation as part of the Air Lead Reduction Act of 1984 (ALRA, 1984, p. 148). Additional data were from the U.S. Geological Survey (USGS, 2005) Lead End-Use table under gasoline additives from 1985 to 1994 to complete the tabulation. These two data sources describe the Pb quantities used in the U.S. during the 1927–1994 era of Pb additive use in gasoline.

2.2. Vehicle traffic and 1982 lead emissions for eight urbanized areas

Vehicle traffic data originating with the Federal Highway Administration were compiled by the Texas Transportation Institute (TTI, 2009). The vehicle traffic data were sorted and tabulated for the following 8 UAs of California: Bakersfield, Oxnard–Ventura, Sacramento, Riverside–San Bernardino, San Jose, San Diego, San Francisco–Oakland, and Metro Los Angeles (see Fig. 1). Fig. 1 indicates the population and location of each UA. It also includes graphs of 1950–1982 population changes (U.S. Census Bureau, 1970, 1990). Population data for the smallest UAs were not available for 1950 and 1960. In general, the UA populations grew at a relatively consistent rate. The main exception is Metropolitan Los Angeles, which between 1950 and 1980 grew more rapidly than the other 8 UAs.

To estimate the quantity of Pb attributable to each of the UAs requires three additional variables, the average fuel economy (mpg), the percent leaded gasoline, and the grams of Pb per gallon.

- average fuel economy for 1982 is from the Federal Highway Administration (U.S. DOTa);
- the quantities of various grades of leaded and unleaded gasoline used in California are from the Ethyl Corporation Yearly Report (Ethyl, 1982); and,
- the annual quantities of Pb per gallon of gasoline are from a published report on the long-term historical trends of Pb in gasoline (Shelton et al., 1982).

Data for each urban area were compiled and tabulated. Daily vehicle miles traveled (DVMT) were summed for freeway and arterial street travel and then multiplied by 365 to obtain the reported vehicle miles traveled (VMT) for 1982. From 1982 VMT the gallons of gasoline were estimated by dividing VMT by the 1982 average of 14.1miles per gallon (U.S. DOTa). The estimated gallons were multiplied by 0.372, assuming the same proportion of leaded gasoline compared with unleaded gasoline for each UA as reported by Ethyl Corporation (1982) for the state of California. Then quantities of Pb were estimated by multiplying the number of gallons of leaded gasoline times 1.14 g per gallon. The estimated Pb quantities were given in metric tons for each urban area in 1982.

According to the U.S. EPA (1986) 75% of the Pb additives were emitted from tailpipes directly into the atmosphere. Particle sizes of

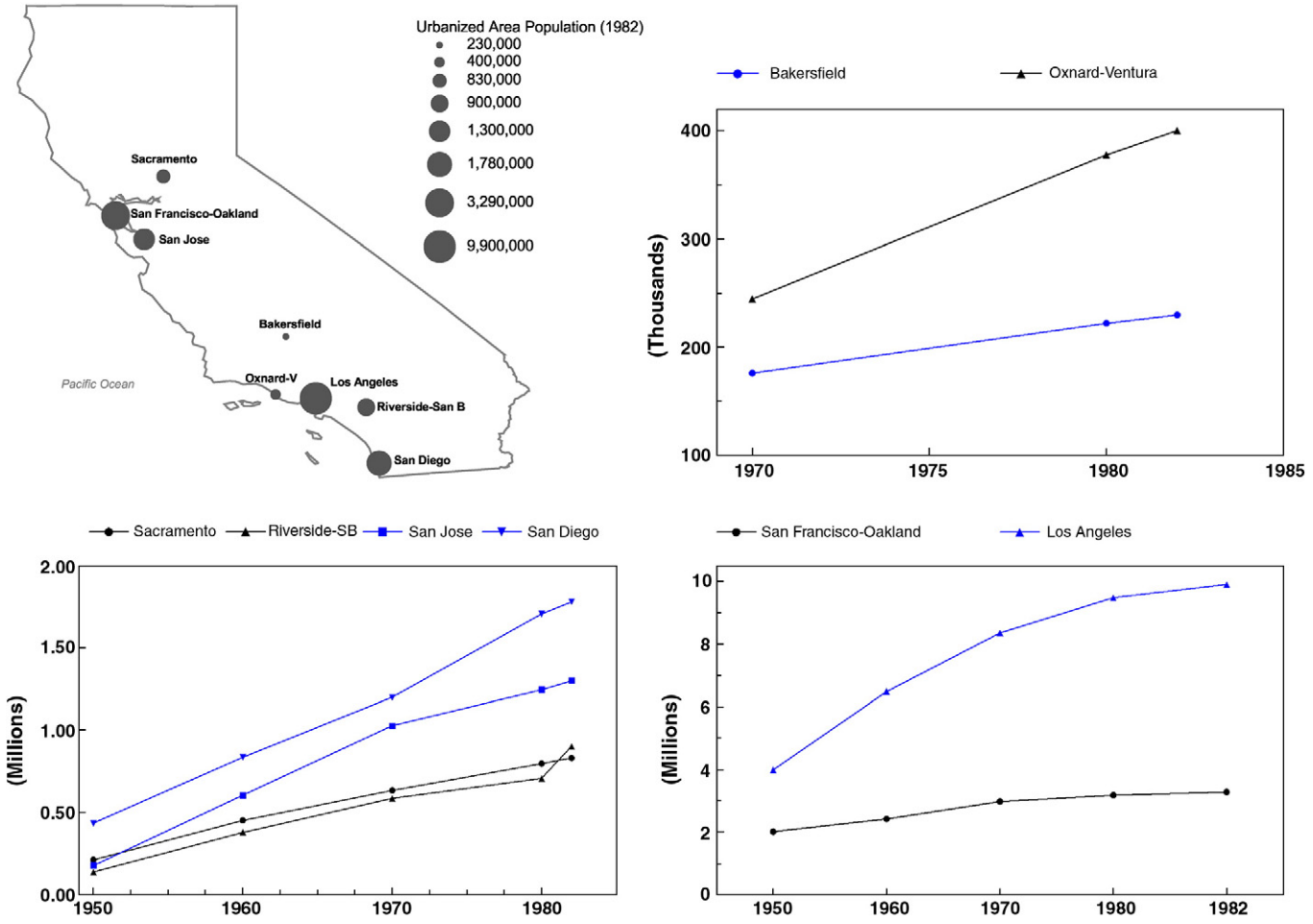

Fig. 1. 1982 Population statistics for eight California urbanized areas where the estimated Pb emissions were developed in this study (U.S. Census Bureau, 1970, 1990). Also, the small graphs show that the population of Los Angeles and Oxnard–Ventura urbanized areas in Southern California grew more rapidly than the other urbanized areas. Because the estimates are based on proportions for 1982, the differences in growth rates tend to inflate the estimates of Pb additives emitted in Los Angeles and Oxnard–Ventura UAs and understate the Pb additives in the other UAs during 1950–1982.

the Pb aerosols were allocated into two categories, >10 μm (40%) and <0.25 μm (35%), and these were also calculated for each urban area (U.S. EPA 1986). Twenty-five percent of the Pb in gasoline remains in the engine as Pb adhering to engine-exhaust surfaces (15%), and Pb remaining in the lubricating oil (10%), and these results are given in Table 1 (U.S. EPA, 1986). The proportion of Pb accounted for by these 8 UAs was 0.63 of the total amount of the 1982 state total of 5180 metric tons for California.

Table 1
Estimated 1982 lead emissions from traffic in eight urbanized areas of California.

Urban area information	Bakersfield Sml	Oxnrd–Vntur Med	Scrmnto Lrg	Rvrsd–Sn Brnrdn Lrg	San Jose Lrg	San Diego Lrg	Sn Frn–Oklnd Vlg	Metro LA Vlg	1982 Sum
Population (1000s)	230	400	830	900	1300	1780	3290	9900	18,630
U.S. rank	76	64	36	33	23	15	8	2	
Urban area (square miles)	70	170	280	450	325	610	800	1830	4535
Popn density (persons/sq mile)	3286	2353	2964	2000	4000	2918	4113	5410	27,044
Peak travelers (1000s)	95	171	344	374	540	739	1296	3990	7549
Freeway DVMT (1000s)	880	3005	5725	6290	11,040	15,070	29,790	72,475	144,275
Arterial streets DVMT (1000s)	1900	2905	8130	7880	9235	11,905	18,895	90,390	151,240
Total DVMT (1000s)	2780	5910	13,855	14,170	20,275	26,975	48,685	162,865	295,515
Total vehicle miles/year (1000s)	1,014,700	2,157,150	5,057,075	5,172,050	7,400,375	9,845,875	17,770,025	59,445,725	107,862,975
Gal/year (1000s) (mpg = 14.1)	71,965	152,989	358,658	366,812	524,849	698,289	1,260,285	4,216,009	7,649,856
Gal/year leaded 0.372 (1000s)	26,771	56,912	133,421	136,454	195,244	259,764	468,826	1,568,355	2,845,747
Grams of Pb at 1.14 g/gal (1000s)	30,519	64,880	152,100	155,558	222,578	296,130	534,462	1,787,925	3,244,151
Metric tons Pb in gasoline in 1982	31	65	152	156	223	296	534	1788	3244
Pb emissions (75%)	23	49	114	117	167	222	401	1341	2433
Med > 10 μm local (metric tons)	12	26	61	62	89	118	214	715	1298
Med <0.25 μm lng rnge trnsprt	11	23	53	54	78	104	187	626	1135
Lead remaining in engine (25%)	8	16	38	39	56	74	134	447	811
Engine and exhaust system (15%)	5	10	23	23	33	44	80	268	487
Lead in the lubricating oil (10%)	3	6	15	16	22	30	53	179	324

The California proportion of U.S. leaded gasoline was 0.372.

2.3. Estimated lead additives in eight California urbanized areas during 1950–1982

Fuel sales for all grades of gasoline in California for 1950–1975 were obtained from Highway Statistics Summary (U.S. DOTb, 1987). The fuel volume by gasoline grade (regular leaded, regular unleaded, premium leaded, and premium unleaded) sold in California during 1975–1982 are listed in the Ethyl Corporation 1982 yearly report (Ethyl, 1982). These gasoline quantities were converted into the amounts of Pb in gasoline by using the data on the historical trends of Pb per gallon of gasoline (Shelton et al., 1982). Further estimates were made by assuming approximately the same proportion of Pb for each California UA between 1950 and 1982 as for 1982. The total amount of Pb additive calculated for California was 412,000 metric tons between 1950 and 1982. The sum of Pb additives estimated for the 8 UAs was 285,000 metric tons. Estimates of the Pb aerosols and Pb remaining in engines and oil are also provided for each UA.

3. Results

Fig. 2 illustrates the trend for the entire six decade period of Pb additives in gasoline as reported by Ethyl Corporation and USGS (ALRA, 1984, p. 148; USGS). The sum of Pb additives in gasoline in the United States from 1927 to 1994 was about 5,368,000 metric tons.

Table 1 lists the 1982 estimated Pb additives used in gasoline by vehicle traffic in 8 UAs of California. The 1982 sum of Pb additives for all 8 UAs is estimated at 3244 metric tons. In addition, the particle sizes of the Pb aerosol are estimated along with the quantity of Pb remaining in the engine system and the lubricating oil (U.S. EPA, 1986).

Fig. 3 is a graph of the annual amount of Pb calculated from fuel sales in California between 1950 and 1982. The U.S. amount of Pb additive calculated for the period from 1950 through 1982 is about 4,639,000 metric tons, or about 86% of the total amount of Pb additive (5,368,000 metric tons) used during the years from 1927 through 1994. The 1950–1982 period brackets the years of most intensive use of Pb additives in gasoline.

Table 2 lists the estimated Pb additives for 8 UAs of California during the 1950 and 1982 period. The tabulation applied the proportions for each UA in 1982 to the eight UAs during 1950–1982. The California total quantity of calculated Pb additive between 1950 and 1982 was about 412,300 metric tons. The amount of Pb additive calculated for the eight UAs is 258,400 metric tons, or 63% of the total Pb additive calculated for California during the same time period. An estimation of the various particle sizes of Pb aerosols and

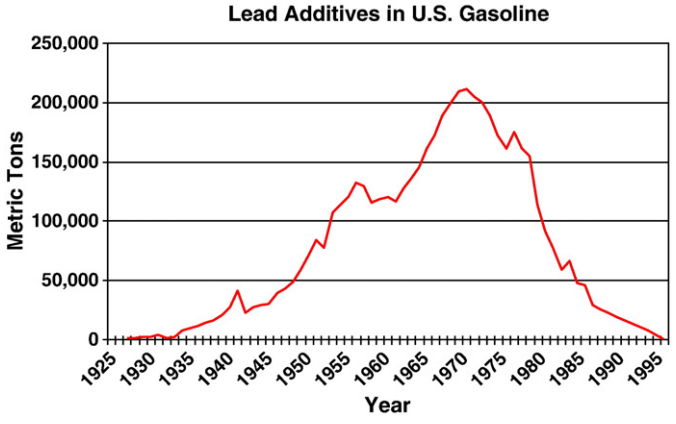

Fig. 2. Total U.S. lead additives in gasoline, 1927–1995, were derived from the proceedings of the U.S. Senate hearings on the Airborne Lead Reduction Act of 1984, S. 2609 (ALRA, 1984) and the U.S. Geological Survey Lead End-Use statistics (USGS, 2005).

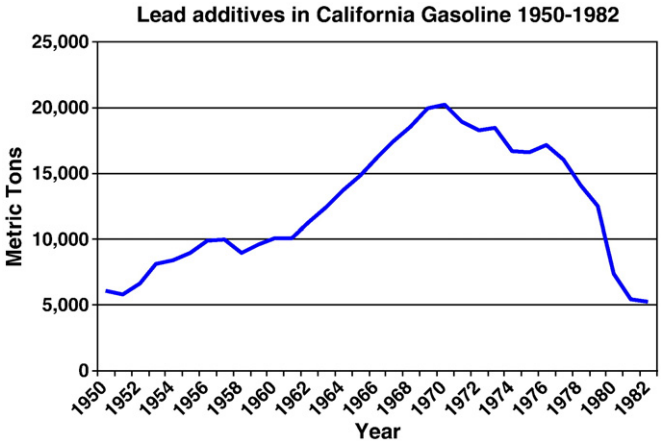

Fig. 3. Lead additives in California gasoline, 1950–1982, were calculated from fuel consumption data from the U.S. Department of Transportation (U.S. DOTb) and the yearly report from the Ethyl Corporation (Ethyl, 1982) using long-term historical trend data on lead additives to gasoline (Shelton et al., 1982). Note that this 32 year span includes the U.S. peak in the use of lead additives.

Pb residues remaining in the engine or crankcase lubricant is also calculated for each of the 8 UAs from the U.S. EPA (1986) data.

4. Discussion

4.1. Quantities of lead in gasoline during 1927–1994

Fig. 2 illustrates the historical U.S. use of Pb additives from Ethyl Corporation plus the USGS data. About 5.4 million metric tons of Pb additives were used in U.S. gasoline beginning in 1927 through 1994. Tetraethyl Pb was touted as safe, with low toxicity as a fuel additive and considered a "Gift of God" by the manufacturers, but in reality Pb additives were an international public health disaster (Kovarik, 2005; Needleman, 1998; Rosner and Markowitz, 1985; Thomas et al., 1999). A rapid increase in Pb additives beginning in 1950 occurred as a result of the combination of a decline of public rail transit systems, a rapid growth in vehicle sales, the rapid expansion of the highway system and restrictions on the use of benzene as an anti-knock additive to fuel (Post, 2007). Before 1950, benzene made up several percent of the fuel. Ironically, because of concerns about the toxicity of benzene, Pb additives replaced benzene to meet anti-knock requirements of the increasing market share of high compression engines. A gradual phase down of Pb additives began in 1975 when EPA introduced health-based language into the regulations (Bridbord and Hanson, 2009). Health considerations required cleanup of engine exhaust and this was achieved by the addition of catalytic converters in the production of new cars. Because the catalysts were rendered useless by Pb, Pb additives in gasoline were removed. When the EPA reduction of Pb additives began to falter in the early 1980s, the U.S. Congress stepped in to ban Pb additives with the passage of the Air Lead Reduction Act of 1984 (ALRA, 1984) which resulted in a rapid phase down on January 1, 1986 and a total ban of Pb additives to gasoline sold for highway use on January 1, 1995. The above facts have direct bearing on the characteristics of Pb in UAs of California.

4.2. 1982 Calculations for eight urbanized areas

Table 1 provides data about the calculated quantity of Pb dust emitted by vehicle traffic within eight California UAs during 1982. The quantities of Pb may be conservative because traffic flow data includes only major freeways and arterial roads and not local traffic between the driveway and arterial roads. Because the estimates are based on 1982 proportions by each UA, the Los Angeles total may be too large and this also means that the totals for the other seven UAs may be too

Table 2
1950–1982 Estimates of the metric tons of lead which were derived from fuel consumed in eight California urbanized areas. The fate of Pb additives after they enter the engine is based on estimates made by the U.S. EPA (1986).

1950–1982 Estimates	Bakersfield	Oxnrd–Vntur	Scrmnto	Rvrsd–Sn Brnrdn	San Jose	San Diego	Sn Frn–Oklnd	Metro LA	City sum	State total
Metric tons Pb in gasoline in 1982	31	65	152	156	223	296	534	1788	3244	5177
Proportion for each city	0.006	0.013	0.029	0.030	0.043	0.057	0.103	0.345	0.627	
Metric tons lead additives 1950–1982	2430	5167	12,113	12,388	17,725	23,583	42,562	142,383	258,351	**412,276**
Pb emissions (75%)	**1823**	**3875**	**9084**	**9291**	**13,294**	**17,687**	**31,922**	**106,788**	**193,764**	**387,527**
Particles med >10 μm local (40%)	972	2067	4845	4955	7090	9433	17,025	56,953	103,341	
Particles med<0.25 μm LRT (35%)	638	1356	3180	3252	4653	6190	11,173	37,376	67,817	
Lead remaining in engine (25%)	**608**	**1292**	**3028**	**3097**	**4431**	**5896**	**10,641**	**35,596**	**64,588**	**129,176**
Engine and exhaust system (15%)	365	775	1817	1858	2659	3537	6384	21,358	38,753	
Lead in the lubricating oil (10%)	243	517	1211	1239	1773	2358	4256	14,238	25,835	

conservative. Also, not all of the Pb emitted by vehicle traffic actually accumulated in the soils of each city. Lead aerosols from undetermined sources drifted large distances and even settled in Greenland and the Antarctic where ice cores disclose a geochemical record of the atmospheric Pb input from industrial development through many centuries (Murozami et al., 1969). For cities, tonnages of relatively course particles of Pb were probably dispersed in a small area, while tonnages of extremely small particles of Pb were scattered across the planet. Small particles of Pb may also collect on buildings, trees, and other vertical surfaces and wash down into the soil (Mielke et al., 1984; Mielke, 1999).

4.3. 1950–1982 Estimates for eight urbanized areas

Fig. 3 illustrates the amount of Pb used in California during 1950–1982. From the data on the fuel consumed each year and the average amount of Pb additive per gallon, the calculated sum of Pb additives is about 412,300 metric tons in California from 1950 to 1982. The total amount of Pb additive used in the U.S. during the same period was about 4,639,000 metric tons; thus, the amount of Pb additive used in California was about 8.9% of the U.S. quantity of Pb additive in gasoline from 1950 to 1982. Table 2 provides the estimated Pb additive for each city for 1950–1982 assuming the same proportions as 1982. The quantity of Pb additive accounted for by these UAs is around 258,400 metric tons of the total Pb additives 412,300, and because the 1982 proportions are assumed, this table accounts for about 63% of the Pb additive in California during 1950–1982. Note that the sum of two of the UAs, Riverside–San Bernardino plus Metro Los Angeles, in the South Coast Air Basin of California for 1950–1982 accounts for about 116,080 metric tons of Pb or nearly 6 times the 20,000 metric tons of Pb that Harris and Davidson (2005) indicated (from 1970 on) for the same region. Fig. 4 is a map of Pb aerosol emissions by the eight UAs of California included in this study. The connection between the legacy of Pb additives in gasoline and the current condition of UAs of California can be evaluated by reviewing soil Pb studies conducted in California.

4.4. Background soil lead concentrations in California

Background soil Pb concentrations are described from several studies. At 11 California Air Force bases the soil Pb concentrations are 5.2 mg/kg (Hunter et al., 2005). For 50 benchmark California soils the

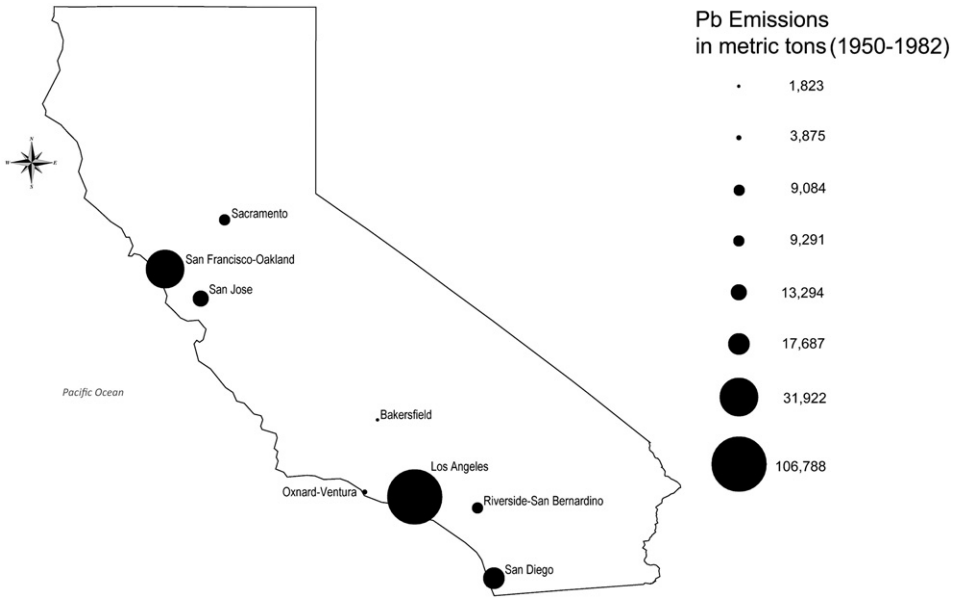

Fig. 4. Estimated lead aerosol emissions in metric tons, 1950–1982, were derived from proportions developed for 1982 that were applied to the calculated Pb additives in grams/gallon (Shelton et al., 1982) times gallons of gasoline sold in California from 1950 to 1975 (U.S. DOTb) and from 1975 to 1982 (Ethyl, 1982). The lead aerosol quantities are 0.75 the total amount of Pb additives in gasoline (U.S. EPA, 1986).

background is 20.6 mg/kg (University of California, 1996). The background was 23 mg/kg for 1300 surface soil samples collected from a 20,000 km^2 study area which includes the western slope of the Sierra Nevada, southern Sacramento Valley and the Coast Ranges of northern California (Goldhaber et al., 2009). Soil samples collected between 1919 and 1933 in the Southern California Air Basin had Pb concentrations of approximately 16 ± 0.5 mg/kg (Page and Ganje, 1970). Compared to UA soils these background soils are remarkably clean and readily accessible to UAs.

4.5. Testable predictions about the legacy of lead additives in California's urbanized areas

The following discussion considers the testable consequences of the legacy of Pb additives in gasoline for 8 UAs of California listed in Tables 1 and 2.

4.5.1. Soil Pb as a function of city size and inner-city vs. outer city location

Given the results of Tables 1 and 2, California UA soils probably have quantities of Pb correlated to city population, i.e., big cities are locations of larger traffic flows and larger emissions of Pb aerosols than smaller cities and towns. This prediction is based on studies conducted on larger and smaller cities and towns of Minnesota which noted that the amount of soil Pb was directly related to the size of the city, and small towns, regardless of age, had less soil Pb than larger towns and cities (Mielke et al., 1984/85, 1989). These city-size studies were replicated in Louisiana and compared with the Minnesota studies, and both studies support the hypothesis that the quantity of vehicle traffic is a major factor in dispersing Pb dust and defining the Pb footprint of urbanized areas (Mielke et al., 1984/85, 1989, 2008; Mielke, 1993). The same city-size characteristics are expected for UAs of California. A study published by the National Research Council in 1972 (NRC, 1972) noted that the Pb content in urban surface soil was 3357 mg/kg at MacArthur Park in Los Angeles, 560 mg/kg at Golden Gate Park San Francisco and 194 mg/kg at Balboa Park, San Diego.

Another characteristic concerning soil Pb quantity is the inner-city vs. outer-city soil Pb differences found in UAs. Laidlaw and Filippelli (2008) and Laidlaw, M.A.S. (Website) reviewed the quantity and distribution of soil Pb in numerous cities in North America, and to date, all North American cities exhibit the same distance decay characteristic of high soil Pb contamination in the inner city and decreasing contamination toward the outer parts of the city as initially identified in garden soils of Baltimore (Mielke et al., 1983). In California, soil studies in the heavily traveled neighborhood of the UCLA married student housing units in Los Angeles ranged between 673 mg/kg and 3633 mg/kg; the same study reports that soil samples from Lancaster, outside of Los Angeles, ranged between 42.5 and 98.4 mg/kg (Johnson et al., 1975). Goldhaber et al. (2009) observed that the UA of Sacramento including the cities of Stockton and Lodi has significantly higher soil Pb concentrations than adjacent rural and less populated areas. Pb concentrations are highest in the eastern Sacramento Valley where the preponderance of UAs and highways are located (Goldhaber et al., 2009). All of the above studies indicate that the major quantities of Pb accumulated in soil are associated with size of, and community location within, UAs of California.

4.5.2. Soil pollution, lead-based paints and vehicle traffic

Given the results of Tables 1 and 2, soils of California UAs probably have soil Pb concentrations that are strongly interrelated with both age of housing units and vehicle traffic. The footprint of Pb in urban soils requires taking into account at least Pb-based paints and Pb additives to gasoline. This hypothesis was tested in Baltimore (Mielke et al., 1983), elaborated on in Minnesota (Mielke et al., 1984, 1989), and further refined for New Orleans (Mielke et al., 1997, 2008). In New Orleans, soil on properties of public housing constructed at the same year with the same materials in the inner-city core and at the outskirts in the city had significantly (p-value < 0.001) different amounts of lead; the inner city (medians 158–386 mg/kg) had two to ten-fold more soil lead than public housing properties located in the outlying areas (medians 37–81 mg/kg) of the city (Mielke et al., 2008). The same study also indicated even larger amounts of lead on soils of residential properties within the inner city (medians 456–707 mg/kg) and the outer city (medians 96–178 mg/kg) (Mielke et al., 2008). Lead-based paint is particularly problematic as a source of pollution in New Orleans because of widespread power sanding. A study of one house was conducted where the paint was scraped and weighed to determine the potential quantity of Pb emanating as Pb dust if it were power sanded (Mielke et al. 2001a). The result was a *one-time* potential release of 7.4×10^9 μg Pb dust which would have been released by power sanding compared to the *annual* emission of 50×10^9 μg of Pb dust per 0.1 mile (0.16 km) from vehicle traffic on a nearby street during the peak (late 1960s–early 1970s; see Figs. 2 and 3) from Pb additives in gasoline (Mielke et al., 2001a).

In California both traffic and building-age related variables are similarly indicated as important variables for predicting soil Pb concentrations. Sutton et al. (1995) analyzed soil samples collected from 358 homes in Oakland, 343 homes in Los Angeles, and 232 homes in Sacramento, and median soil concentrations were 880 mg/kg in Oakland, 190 mg/kg in Los Angeles and 222 mg/kg in Sacramento; Sutton et al. (1995) also observed that homes built before 1920 were 10 times more likely to have soil Pb content ≥500 ppm compared to post-1950 homes; in Oakland, soil Pb concentrations exceeded 1000 mg/kg at 46% of the homes. A description of the location of the homes in each city is missing, but older homes are commonly located in inner-city communities while newer homes are located in communities toward the outskirts of UAs.

A review of California studies indicates that compared with background soil Pb away from urban areas, soils in UAs are highly contaminated by highway sources of Pb. In 2002, after Pb additives to gasoline had been completely phased out, Lejano and Ericson (2005) analyzed soil around Pacoima, California and found that both total and bio-available Pb were markedly higher in areas close to major highways. Similarly, elevated soil Pb occurred within a residential community in the proximity of Interstate 880, Alameda County, California (Teichman et al., 1993). Soils were collected from the yards of homes in communities adjacent to the freeway and within a one mile (1.6 km) radius; the possibility of Pb-based paint contributing to the contamination was minimized by collecting samples at least 20 ft (>6 m) from the homes. The soils closest to the highway contained amounts of Pb exceeding California's and EPA's criteria for hazardous waste, or >1200 mg/kg (Teichman et al., 1993). Wu et al. (2010) collected 550 surface soil samples from south central Los Angeles and found that mean total and bio-available Pb concentrations were highly correlated ($r = 0.96$); Pb concentrations near freeways and major arterials were significantly higher than soils collected at other locations.

Vehicle traffic continues to impact Pb loading of street dust. In addition to the past usage of Pb additives that have accumulated in UAs, Pb-based wheel weights used to balance tires contribute to Pb dust loading. Wheel weights are prone to dislodgement and on pavement they are ground into fine particles by the pounding forces of traffic (Root, 2000; U.S. EPA NLFWWI). California has passed legislation that was signed into law to eliminate Pb in wheel weights on cars intended for sale in California by July 31, 2009 (California lead-based wheel weight ban, Senate Bill #757). Pb however, still remains as a legacy of pollution associated with streets and highways.

4.5.3. Soil lead as a source of exposure

Given the quantities of Pb shown in Tables 1 and 2, Pb dispersed and settled in soils of California UAs probably exposes children.

Children are exposed to soil Pb by dust being tracked into homes on shoes (Hunt et al., 2006) and family pets and via resuspension and deposition of Pb contaminated urban soil dust which penetrates interiors of homes and settles onto contact surfaces (Clark et al., 2004; Layton and Beamer, 2009; Laidlaw and Filippelli, 2008). Children are then exposed through hand-to-mouth activity (Ko et al., 2007). Soil Pb concentrations have been observed to be associated with children's blood Pb concentrations using multiple study designs—cross-sectional, ecological spatial, ecological temporal, prospective soil removal and isotopic (Filippelli et al., 2005; Laidlaw and Filippelli, 2008). Lead in soil is at least co-equal to Pb-based paint as an explanation for the urban pattern of Pb exposure of children (Mielke and Reagan, 1998). Children's hands at childcare centers were tested with wipes before and after outdoor play, and they had more Pb after playing outside than after playing inside (Viverette et al., 1996; Nielsen and Kristiansen, 2005). At private inner-city childcare centers the amount of Pb per hand exceeded the 6 μg Total Tolerable Daily Intake (TTDI) of Pb by a factor of 5 or more; in public Head Start childcare centers, where bare soil on play areas is covered with rubber mats, children's hands did not indicate Pb differences before and after outside play (Viverette et al., 1996; Nielsen and Kristiansen, 2005). Furthermore, a soil surface sampler (PLOPS) revealed that the Pb loading on the bare surface of soil containing 400 mg/kg, the EPA standard for Pb, exceeds the current guideline of Pb loading of 40 μg/ft^2 on interior floors by a factor of about 35 (Mielke et al., 2007b). Soil Pb on the play areas of elementary public schools follow the same trend as soil Pb in the entire city; however, soils on school grounds are significantly less Pb contaminated than soils of neighboring residential properties where the most vulnerable preschool children probably play before they attend school (Higgs et al., 1999).

Finally, vehicle interiors may also be a source of Pb exposure. In a case reported of car contamination from clothes of employees who work with Pb, Pb dust collected in vehicle upholstery and carpets may exist as another probable source of children's Pb exposure (Bernier et al., 2009; Yiin et al., 2002).

4.5.4. Association between soil lead and blood lead

Given the results shown in Tables 1 and 2, blood Pb of California children probably respond directly to size of UAs and community location within a given UA. This and other predictions regarding blood Pb require obtaining full spectrum blood Pb surveillance rather than blood Pb results truncated at an arbitrary level (such as 15 μg/dL) as currently occurs in California. In Minnesota, the amount of Pb in the soil of a community was found to be strongly associated with blood Pb of children living in the same community, and this relationship was further refined for Louisiana (Mielke et al., 1989, 1997, 1999, 2007a). Levin et al. (2008) state that blood Pb levels increase 1–5 μg/dL for every 1000 mg/kg increase in soil Pb. However, the blood Pb response of children to soil Pb is curvilinear in New Orleans, LA (Mielke et al., 1997, 1999). Johnson and Bretsch (2002) also observed a similar curvilinear relationship between soil Pb and children's blood Pb in Syracuse, NY. The most recent New Orleans urban soil Pb and blood Pb study shows the following results: Below 100 mg/kg soil Pb children's blood Pb response is steep at 1.4 μg/dL per 100 mg/kg, while above 300 mg/kg soil Pb children's blood Pb response is a gradual 0.32 μg/dL per 100 mg/kg (Mielke et al., 2007a). Similar associations between soil Pb and blood Pb responses of children are expected in UAs of California.

In Los Angeles, Macey et al. (2001) concluded that proximity to transportation corridors was consistently the strongest indicator of environmental Pb exposure, while median home values were significantly and inversely associated with elevated blood Pb levels. In the UA of Sacramento, Deocampo and Orr (2006) observed that soil Pb concentrations ranged from 15 mg/kg (background) to over 1500 mg/kg and that the spatial distribution of soil Pb concentrations showed a possible relationship with children's blood Pb concentrations.

4.5.5. Soil lead and seasonality

Given the results shown in Tables 1 and 2, blood Pb seasonality in California UAs will probably respond directly to climatic variables affecting Pb dust accumulated in each of the UAs. The Pb dust within major cities is so ubiquitous that seasonal weather patterns impact the blood Pb of the childhood population. For example, in Mexico City, Rosas et al. (1995) observed that during rainy seasons of the year, PM 10 dust was settled and atmospheric Pb concentrations were lower; during seasons with low rainfall PM10 and atmospheric Pb concentrations were higher. Laidlaw et al. (2005) extended this observation to include blood Pb and observed that during droughty periods when soil is dry and dusty, blood Pb increases; during rainy periods when soil is wet and dust is settled, blood Pb decreases. The suspended Pb dust apparently settles out and gradually recontaminates clean soil (Mielke et al., 2006b). Many studies indicate that urban Pb contaminated soils are being resuspended in the summer and autumn when evapotranspiration is at a maximum.

Average monthly blood Pb (BPb) values of children from urban areas tends to increase significantly in summer and autumn months (Haley and Talbot, 2004; Laidlaw et al., 2005; Yiin et al., 2000). Early work by Mielke et al. (1992), Johnson et al. (1996), and Johnson and Bretsch (2002) suggested that blood Pb seasonality may be related to the interaction between climate and Pb contaminated soils. Recent research strongly supports the relationship between soil resuspension and exposure and children's blood Pb levels. Laidlaw (2009) attempted to determine if the seasonal resuspension of soils into the atmosphere was related to seasonal changes in atmospheric Pb concentrations. Laidlaw (2009) tested the hypothesis that atmospheric Pb and atmospheric soil concentrations obtained from the Interagency Monitoring of Protected Visual Environments (IMPROVE, 2007) exhibit statistically significant correlations in Detroit, Birmingham and Pittsburgh. Results indicate that atmospheric soil and atmospheric Pb were correlated in Detroit between November 2003 and July 2005 ($r=0.47$, $p=<0.001$); in Pittsburgh between April 2004 and July 2005 ($r=0.40$, $p<0.001$); and in Birmingham between May 2004 and December 2006 ($r=0.35$, $p<0.001$). Laidlaw concluded that soil and atmospheric Pb follow seasonal patterns with highest concentrations during the summer and/or autumn, and that atmospheric Pb and atmospheric soil concentrations are correlated due to resuspension of urban Pb contaminated soils. Laidlaw (2009) suggested that this Pb may be causing seasonal variations in children's blood Pb levels. In Milwaukee, Wisconsin, Havlena et al. (2009) observed that blood Pb levels followed a seasonal pattern with peaks in the summer and autumn. They also observed that particulate matter less than 2.5 μm (PM2.5) correlated with the seasonal variation in 10 month old children's blood levels, and suggested that the Pb in the PM2.5 was causally related to seasonal variations in children's blood Pb levels.

The assumption that soil Pb is being resuspended and is responsible for a large portion of the Pb in the atmosphere is supported by isotopic analysis of atmospheric Pb in Yerevan Armenia (Kurkijan et al., 2002) which indicated that following elimination of the use of Pb in gasoline, 75% of atmospheric Pb in the Yerevan atmosphere was derived from resuspended soil. Similarly, Kamenov (2008) analyzed Pb isotopic ratios of teeth in Sofia Bulgaria and found that remarkable isotopic similarities between the teeth and the Pb additive to gasoline in the local soils and concluded that soil and/or soil-borne dust inhalation and/or ingestion are the most probable pathway for incorporation of soil Pb in the local population. Kurkijan et al.'s (2002) and Kamenov's (2008) observations are supported by Pingitore et al. (2009) who used synchrotron-based XAFS (x-ray absorption fine structure) to quantify the Pb species in the air of El Paso. Pingatore found that Pb-humate was the dominant form of Pb in contemporary El Paso air and that Pb-humate is the major Pb species in El Paso soils. Pingitore et al. (2009) concluded that the soil must be the dominant source, and it was being resuspended into the atmosphere.

Lankey et al. (1998) based on 1989 data, estimated that leaded gasoline use was responsible for approximately 50% of the overall lead emissions. Harris and Davidson (2005) calculated that the Pb particles that were deposited in the Southern California Air Basin (SOCAB) during the years of Pb additives in gasoline use are being resuspended into the atmosphere and responsible for generating approximately 54,000 kg of airborne Pb each year. The Harris and Davidson (2005) study used an average soil concentration of 79 mg/kg as an input into their resuspension model, while Wu et al. (2010) has calculated that the median soil concentration in Los Angeles is 180 mg/k. Thus Harris and Davidson's (2005) SOCAB Pb resuspension estimate may be conservative. Nevertheless, Harris and Davidson (2005) concluded that soil contamination contributes most of the total airborne Pb currently measured in the SOCAB and is likely to continue to do so for many years. Young et al. (2002) studied resuspended roadside soils and Pb enriched smelter soils and observed that the Pb in the particulate matter less than 10 μm (PM10) was enriched by 5.36–88.7 times compared with uncontaminated California soils.

Seasonal weather patterns differ markedly between southern and northern California and these differences are expected to influence children's blood Pb responses. Sabin and Schiff (2008) took measurements of metal dry deposition fluxes weekly on a north–south transect along the coast of southern California between Santa Barbara and San Diego over a four-month period during summer and fall 2006. Lead fluxes varied between 0.52 and 14 μg/m^2/day with the highest Pb fluxes occurring at the San Diego and Los Angeles sites. They also commented that model estimates and measurement data in Los Angeles have found metal dry deposition flux rates which may increase during Santa Ana wind conditions by as much as factors of two to eight (Sabin and Schiff, 2008). Atmospheric metal concentrations at three sites in Santa Barbara and one site in Riverside showed evidence of seasonal variations whereby outdoor concentrations of metals were higher in the warmer months than in the cooler months (Polidori et al., 2009). Similarly, children's blood Pb in South Central Los Angeles ($N=3679$) from 1991 to 1994 displayed a significant seasonal effect with the lowest blood Pb during late winter and early spring, and the highest blood Pb levels occurring in summer (Rothenberg et al., 1996).

4.5.6. School performance and soil lead

Lead is the most intensively studied toxin, and the neurotoxic impact of childhood Pb exposure is strongly associated with societal problems that are extremely costly to society including learning deficits, socialization and violent behavior (Chandramouli et al., 2009; Muennig, 2009; Nigg et al., 2008; Reyes, 2007; Zahran et al., 2009). Given the results shown in Tables 1 and 2, California school children probably have school performance responses that are associated with the amount of Pb dust accumulated in various communities of UAs. The bases for this prediction are studies on tooth Pb and school performance by Needleman et al. (1979) followed up by empirical studies conducted in Minnesota and New Orleans. The hypothesis that the amount of Pb in soils of a community is associated with the performance of 4th grade children attending local schools was first hinted at by the observation in Minnesota of an association between dropout rates of high school students and city size (Mielke et al., 1989). The New Orleans studies refined the research to include the association between school achievement test scores and community soil Pb and other metals (Mielke et al 2005b; Mielke and Berry, 2007, pp 116–123), and then between blood Pb and achievement test scores as an indicator of neurotoxicity at New Orleans attendance district schools (Zahran et al. 2009). Zahran et al. (2009) support Landrigan etal. (2002) and Stefanak et al. (2005) concerning the insidious costs that environmental Pb imposes on urban society. The finding also supports other reports (Chandramouli et al., 2009; Nigg et al., 2008). Patterson (1980, pp 271–272) expressed the problem as follows: "Extrapolating from present information, …probably… it will be shown in the future that average American adults experience a variety of significant physiological and intellectual dysfunctions caused by long-term chronic lead insult to their bodies and minds which results from excess exposures to industrial lead that are five hundred-fold above natural levels of lead exposure, and that such dysfunctions on this massive scale may have significantly influenced the course of American history."

Other important findings regarding soil pollutants and health are the correlations between multiple metals first described in Baltimore and also noted in New Orleans (Mielke et al., 1983, 2000). Lead, zinc, cadmium, manganese, chromium, nickel, vanadium and copper are strongly correlated with each other in soil, and the strongest correlations are among Pb, Zn, Cu, and Cr respectively (correlation coefficients above 0.8) (Mielke et al., 2000). The extremely strong correlation between Pb and Zn is probably because both metals are connected with vehicle traffic (Pb in gasoline and Zn in tires). Mixtures of multiple metals and various poly aromatic hydrocarbons (PAHs) are also elevated in soils in the inner city compared with soils in outlying communities (Mielke et al., 2001b; Wang et al., 2004). The toxicological impact of chemical mixtures is poorly understood but it probably influences the toxic characteristics of inner-city soils compared with soils in outlying communities of New Orleans, and these characteristics suggest a chemical basis for issues regarding health disparities and environmental justice (Campanella and Mielke, 2008; Mielke et al., 2004). Thus, school performance response by California children will probably be complicated by environmental chemistry characteristics and seasonality differences within the eight UAs included in this study.

4.5.7. Treating the urban lead pollution problem

Currently U.S. exposure reduction efforts are focused on interior residential Pb dust, and especially on Pb-based paints (Dixon et al., 2009; Gaitens et al., 2009; Levin et al., 2008; Roberts et al., 1999). Given the results of Tables 1 and 2, prevention of Pb exposure in California UAs with emphasis on Pb-based paint alone will not successfully curb low blood Pb exposures (i.e. 5–10 μg/dL or less) among children. In California a soil Pb standard of 80 mg/kg is proposed for residential properties to protect children from Pb (California 2009). This Pb standard is the same as empirically derived in New Orleans which assumed that the policy is to protect most children from a blood Pb exposure ≥10 μg/dL (Mielke et al., 1999). If the policy is changed to prevent exposure of most children to a blood Pb<5 μg/dL or lower, then the soil Pb standard must be reduced to substantially less than 80 mg/kg (Zahran et al., 2010). Because of the inherent size of the reservoir, soil Pb has been described as the "elephant in the playground" (Filippelli and Laidlaw, 2010). Soil mapping assists with developing priority for treatment and an empirical means for visualizing the environmental quality of any given city (Mielke, 2002). Some soil Pb mapping has been reported for UAs of California, but the surveys are not as extensive as maps of New Orleans (Mielke, 1991, 1994; Mielke et al., 2005a) or Syracuse (Griffith et al., 2009). California urban survey data are probably not of sufficient density to guide remediation efforts.

Examples of treating contaminated urban soil as a method for preventing exposure and reducing blood Pb began in Minneapolis (Mielke et al., 1992) and was advanced in New Orleans by using the soil Pb map as a guide (Mielke, 1994). Fresh sediments of the Mississippi River flow by the city at the average rate of 300 tons per minute, and they are remarkably clean (~5 mg Pb/kg) and therefore it is a valuable natural resource to use for covering contaminated soils in the city (Mielke, 2005; Mielke et al., 2006a,b). Precedence for a national clean soil effort has been established by Norway to prevent exposure to accumulated Pb dust and other pollutants in urban environments. Norway's clean soil program conducts testing and cleanup of contaminated soils at all childcare centers, elementary schools and parks in the ten largest cities of the nation (Ottesen et al.,

2008). The scientific basis for developing the program was motivated in part by the mapping and statistical analysis applied to New Orleans research on soil Pb and children's health response (Ottesen et al., 2008).

5. Conclusions: emerging precautionary approaches to primary exposure prevention

Lead is a well known toxin, and its neurotoxic impact on children is strongly associated with problems that are extremely costly to society, including learning deficits, socialization, violent behavior and other health problems. Urban soil geochemistry research began with a map and statistical analysis of the clustering of high Pb concentrations in garden soils of inner-city Baltimore and the method has been developed into a tool for predicting the exposure of children to Pb. Emerging issues include finding the ways and means to reduce the continuing influence of Pb dust on children's health and wellbeing. Young children have no possibility for creating Pb-safe environments for themselves—this is an adult responsibility. There are many ways to make the environment safer for children. Currently, following Norway's precedence, an exterior mapping and clean soil program at childcare centers, elementary schools and playgrounds provides a path toward primary Pb exposure prevention for young children. By following the same precedence, California's children would benefit from an urban soil Pb mapping and clean soil program.

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Chapter 3 - Paper 2:*"Lead (Pb) legacy from vehicle traffic in eight California urbanized areas: continuing influence of Pb dust on children's health."*

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Review

Estimation of leaded (Pb) gasoline's continuing material and health impacts on 90 US urbanized areas

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ABSTRACT

The subject of this paper is lead (Pb) additives in gasoline and their material and health impact from Pb dust inputs into 90 US urbanized areas (UAs). The mass of Pb additives for 90 UAs as a total of the US Pb additives in 1982 were estimated from vehicle travel, vehicle fuel economy (miles/gallon), ratio of leaded to unleaded fuel, and Pb/gallon. About 500 billion (10^9) miles of travel in 90 UA's during 1982 account for ~18,000 metric tons (MT), or nearly 30% of the US Pb additives in 1982. Applying the 1982 proportions to the 90 UAs for 1950 through 1982 fuel sales by state accounts for ~1.4 million MT Pb of the US national total of 4.6 million MT during the same years. Fates of Pb additives in engine systems were used to calculate Pb aerosol inputs into the 90 UAs. The inputs range from 100's to more than 100,000 MT of Pb depending on a given UA's traffic flow patterns. Soils are the reservoir of urban Pb dust. The median background soil Pb for the US is 16.5 mg/kg (range 10.3 to 30.1 mg/kg), and less by an order of magnitude or more than soil Pb within larger UAs. Recognizing the US input of massive gasoline Pb additives into UAs assists with comprehending soil Pb differences between large and small UAs, inner and outer areas of UAs, health disparities, and school achievement issues within UAs. The findings underscore the need for controlling accumulated exterior urban Pb dust from gasoline additives along with paint sources that have accumulated in soil to meet the goal of primary childhood Pb exposure prevention.

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Abbreviations: ft^2, 0.09290304 m^2 or 929 cm^2; 40 μg/ft^2, 431 μg/m^2; DVMT, Daily vehicle miles of travel; MT, Metric tons; NHANES, National Health and Nutrition Examination Survey; US gallon, 3.79 L; US miles, 1.61 km; VMT, Vehicle miles traveled per year.

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1. Introduction

The subject of this paper is lead (Pb) additives to gasoline and their material and health impact from Pb dust inputs into 90 US urbanized areas (UAs). The history of the discovery, early industrial accidents, opposition to, commercial use, and the January 1, 1986 rapid phase out of ~90% of the Pb additives to gasoline are well described in the literature (Bridbord and Hanson, 2009; Kovarik, 2005; LaBelle et al., 1987; Needleman, 1998; Nriagu, 1990; Rosner and Markowitz, 1985). About the same mass (5–6 million MT) of anthropogenic Pb was used in the commercial production of nonpaint Pb additives to gasoline as in paint production (Mielke and Reagan, 1998). The World Health Organization emphasizes accumulated Pb dust from paint and nonpaint sources in soil as a major concern for children's health (Louis et al., 2006). US policy emphasizes lead-based paint as the major concern for children's health and indicates that nonpaint sources are "insufficiently characterized... [and] ... often underestimated" (Levin et al., 2008). In 1978, paint sources of Pb were restricted to 600 mg/kg, and in 1995 the ban was completed on Pb additives to gasoline for US highway travel (Levin et al., 2008; US EPA, 1996a). The use of Pb-based paint on older buildings is the subject of intense scrutiny. Estimation of the magnitude of additives in gasoline as a nonpaint source of Pb inputs into urban environments must also be scrutinized to comprehend the impact of essentially equal masses of Pb in paint and gasoline on the environment and health.

The purposes of this paper are: 1) to estimate the mass of gasoline Pb aerosols dispersed into US urbanized areas from 1950 through 1982, 2) to review studies concerning US background soil Pb and soil Pb in US urbanized areas, and 3) to characterize the environmental health impact that the input of Pb dust may have on various sized UAs.

2. Methods

First, the mass of gasoline Pb additive from 1950 to 1982 were calculated from state fuel sales and compared with data from the Ethyl Corporation (US Senate, 1984). Second, the proportions of Pb additives used in 90 US urbanized areas were calculated for 1982 from vehicle travel data and public records. Third, the proportion of the Pb additives in each UA in 1982 were applied to state by state fuel consumption records from 1950 to 1982 to derive the quantities of Pb used in the 90 UAs. Finally, a review of soil Pb and health studies was conducted to assist with characterizing the environment and health effects of gasoline Pb additives on the 90 UAs.

2.1. Mass of gasoline lead additives in the US 1950–1982

Fuel sales by state for all grades of gasoline for 1950–1975 were obtained from Highway Statistics Summary (US DOT, 1987). The fuel volume by gasoline grade (regular leaded, regular unleaded, premium leaded, and premium unleaded) sold in each state from 1975 to 1982 were obtained from the Ethyl Corporation (1982) yearly report. The gasoline quantities were converted into the amounts of Pb from data on the historical trends of grams of Pb per gallon for the various grades of gasoline (Shelton et al., 1982).

2.2. 1982 Traffic lead emissions for 90 urbanized areas

Vehicle traffic data is collected by every state using guidelines from the US Department of Transportation, Federal Highway Administration (US DOT 1999). These data are an essential requirement for setting priorities for taxpayer expenditures of highway construction and maintenance in the US. Vehicle traffic data originating from the Federal Highway Administration are compiled by the Texas Transportation Institute (TTI, 2009). The TTI vehicle traffic data were sorted and tabulated for 90 urbanized areas. To estimate the quantity of Pb attributable to each of the urbanized areas, the following data were assembled for 1982: vehicle traffic mileage for 1982 for 90 UAs (TTI, 2009), fuel efficiency in miles per gallon or mpg (US DOT), percentage of various grades of gasoline (Ethyl, 1982), and grams of Pb per gallon of leaded fuel for 1982 (Lewis, 1985). All of these data components for 90 UAs were available only for 1982.

Lead dust for each urbanized area were estimated as follows: daily vehicle miles traveled (DVMT) were summed for freeway and arterial street travel and then multiplied by 365 to obtain the annual vehicle miles traveled (VMT) for 1982. The 1982 gallons of gasoline were estimated by dividing the annual VMT by the 1982 average miles per gallon (14.1 mpg). The volume of gasoline was multiplied by state proportions of leaded gasoline as reported by the Ethyl Corporation (1982). In the cases where UAs are linked with multiple states, the averages of the combined state proportions of leaded gasoline were used. Then mass of Pb for each UA were estimated by multiplying the number of gallons of leaded gasoline times 1.1 g per gallon (Lewis, 1985). Proportions were assigned by dividing the mass of Pb additives from vehicle traffic of each UA by the total mass of Pb additives for the nation in 1982 (Mielke et al., 2010; US Senate Hearings, 1984).

2.3. Estimated Pb additives and Pb aerosols into 90 UAs 1950–1982

Assuming approximately the same proportion of Pb for each of the 90 UAs in 1950–1982 as for 1982, estimates of total Pb were made for each UA. The fates of Pb additives in gasoline were based on information from the US EPA (1986). According to the EPA, 75% of the Pb additives were emitted as exhaust directly into the atmosphere. Particle sizes of the Pb aerosols were allocated into two categories, >10 micrometers (μm) (40%) and <0.25 μm (35%), also 25% of the Pb in gasoline remained in the engine and adhered to

engine or exhaust surfaces (15%), and the remaining Pb (10%) entered the lubricating oil (US EPA, 1986).

3. Results

3.1. Annual mass of US Pb gasoline additives from 1950–1982

Fig. 1 is a graph of the annual masses of Pb in metric tons (MT) calculated from fuel sales in the US between 1950 and 1982 along with the quantities of Pb additives reported by the Ethyl Corporation for the same time period at the Senate Hearings on the Airborne Lead Reduction Act of 1984, S.2609 (US Senate, 1984). The total amount of US Pb additive calculated from state fuel consumption records between 1950 and 1982 is 4.64 million MT. This compares with 4.61 million MT reported for 1950 through 1982 by the Ethyl Corporation (US Senate, 1984); the correlation coefficient is 0.981 (P-value 0.74×10^{-11}) between the two data sets. The close match between the quantities of Pb additives calculated from fuel sales and the amounts reported at the US Senate Hearings by the Ethyl Corporation provide confidence in the calculation methods. The 1950 through 1982 period accounts for about 86% of the total mass of 5.37 million MT Pb used as a gasoline additive during the years from 1927 to 1994 and brackets the most intensive years of commercial sales of gasoline with Pb additives.

3.2. 1982 lead estimates for 90 urbanized areas

The 1982 proportions of Pb for the 90 urbanized areas (18,222 MT) were nearly 30% of the total amount of Pb (61,526 MT) sold for the US consumption in the same year. The US EPA (1986) fate of Pb in the engine including the particle sizes of the Pb aerosols along with the quantity of Pb remaining in the engine system and the lubricating oil were also calculated for the 90 UAs.

3.3. 1950–1982 estimates for ninety urbanized areas

Using the same proportions for each UA as calculated for 1982, the amount of Pb additive for the 90 urbanized areas for 1950 through 1982 is 1.37 million MT or about 30% of the 4.64 million MT Pb additive total in the US calculations of the particle sizes of Pb aerosols and Pb remaining in the engine or crankcase lubricant are given in parentheses.

Table 1 is a summary of the total quantity of Pb in MT (and in parenthesis the estimated mass of Pb aerosol emitted) for 1950 through 1982.

Table 2 lists the rank by metric tons of Pb additive estimated for each UA from highest (1) to lowest (90) for 1950 through 1982, and categorizes each of the UAs into quartiles according to the metric tons of Pb additives and aerosol emissions given in Table 1. The approximate location, rank of each of the UAs and symbols for each quartile are illustrated in Fig. 2.

4. Discussion

4.1. Limitations

Estimates for the Pb additives in 1982 were calculated from vehicle traffic data characteristics for each UA as a proportion of the total amount of Pb additives in 1982. The proportions were then applied to total Pb additives calculated from state by state gasoline sales from 1950 to 1982. The main limitation is a consequence of the assumption that the 1982 proportions of Pb for each UA are appropriate to apply to the 1950 through 1982 period for each of the 90 UAs. For example, Los Angeles UA grew more rapidly than most other UAs from 1950 to 1982 (Mielke et al., 2010). As a result, by applying the 1982 proportions to the entire 1950 through 1982 period, the estimates of Pb for the 1950s, 1960s and 1970s would be overestimated for the Los Angeles UA. An overestimate of Pb additives for the Los Angeles UA would result in an underestimate of Pb for the other UAs. However, the 1950 through 1982 Pb additive estimates shown in Tables 1 and 2 represent ranges of Pb additives and Pb aerosols inputs within various sized urbanized areas. These results are a reasonable estimate of the mass of Pb from gasoline associated with each UA and provide a beginning point for characterizing the effects of the inputs of gasoline Pb additives on the various UAs.

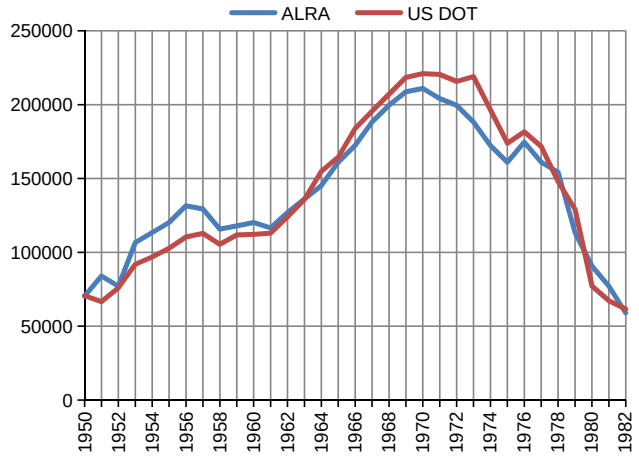

Fig. 1. Comparisons between Pb additives in gasoline by methods using state by state fuel consumption data from 1950 to 1982 (US DOT) and Ethyl Corporation data presented at the Airborne Lead Reduction Act (ALRA) hearings conducted by the US Senate in June, 1984 (US Senate 1984). All numbers on the left axis are in metric tons per year.

Table 1
Estimated mass of lead additives (and aerosol emissions) by a group of urban areas and percentiles for 90 urbanized areas from 1950 to 1982. See also Fig. 2. (In metric tons).

Percentiles	1st group	2nd group	3rd group	4th group
	Additives (aerosols)	Additives (aerosols)	Additives (aerosols)	Additives (aerosols)
Min	17,686 (13,265)	8403 (6302)	4662 (3497)	608 (456)
25%	24,859 (18,644)	9506 (7130)	5374 (4031)	1907 (1430)
50%	31,601 (23,701)	11,737 (8803)	5709 (4282)	3080 (2310)
75%	44,821 (33,616)	14,310 (10,733)	6802 (5102)	4055 (3041)
Max	149,938 (112,454)	16,623 (12,467)	8108 (6081)	4570 (3428)
N =	23	22	22	23

4.2. Characterization of the impact of gasoline Pb additives

General characterization of Pb additives must begin by recognizing the massive quantity of Pb mined and smelted for commercial use in gasoline. Lead is an element and when emitted as an aerosol it persists in the environment. Lead accumulates mainly in the loose surface materials (i.e. pedosphere) such as soils and/or stream and marine sediments. The Pb accumulated in soil will remain for hundreds of years (LaBelle et al., 1987). Han et al. (2002) estimated that by the year 2000, the cumulative global industrial production of Pb was about 235 million MT. Thus, during the short span of about 100 years, US paint and gasoline additives accounted for a combined total of 10–12 million MT of Pb, or about 5% of the several thousand year global history of anthropogenic Pb production.

4.2.1. Pb aerosol particle sizes

Characterization of gasoline Pb additives requires a description of the physical characteristics of Pb aerosol particles. EPA (1986) estimated that 75% of Pb additives were emitted as exhaust. The tonnages of the relatively large >10 μm particles of Pb probably settled locally, especially in the urbanized areas where Pb in soils are elevated adjacent to roadways and decrease with distance away from the roadway (Laidlaw and Filippelli, 2008). Thirty-five percent of the particles were <0.25 μm size. The bulk of the smallest aerosol particles were emitted in the ultra-fine particle size, or less than 0.1 μm diameter (Blom et al., 2000). The tonnages of the ultra-fine particles of lead were dispersed throughout the planet and deposited in oceans and on glaciers such as Greenland and the Antarctic where ice cores disclose a geochemical record of the atmospheric lead input from many centuries of industrial production (Han et al., 2002; Murozami et al., 1969).

However, a portion of the ultra-fine particles of Pb were also retained in urbanized areas. Because of their density, ultra-fine Pb particles tend to pierce through the boundary layer of air surrounding

Table 2
Urbanized areas listed and grouped by rank of lead additives during 1950–1982. See Fig. 2 for locations numbered by rank for the contiguous states, not including Alaska and Hawaii.

Group 1	Group 2	Group 3	Group 4
1 LA-Long Bch-Santa Ana CA			68 New Haven CT
2 New York-Newark NY-NJ-CT	24 Baltimore MD	46 Bridgeport-Stamford CT-NY	69 Columbia SC
3 Chicago IL-IN	25 Indianapolis IN	47 Orlando FL	70 Albany-Schenectady NY
4 Detroit MI	26 Milwaukee WI	48 Buffalo NY	71 Rochester NY
5 Dallas-Ft Worth-Arlington TX	27 Virginia Beach VA	49 Omaha NE-IA	72 Allentown-Bethlehem PA-NJ
6 San Francisco-Oakland CA	28 San Antonio TX	50 Dayton OH	73 Toledo OH-MI
7 Houston TX	29 Oklahoma City OK	51 Hartford CT	74 Colorado Springs CO
8 Philadelphia PA-NJ-DE-MD	30 Riverside-San Brnardino CA	52 Austin TX	75 Poughkeepsie-Newburgh NY
9 Boston MA-NH-RI	31 Cincinnati OH-KY-IN	53 Richmond VA	76 Spokane WA
10 Miami FL	32 Sacramento CA	54 Raleigh-Durham NC	77 Corpus Christi TX
11 Atlanta GA	33 Tampa-St. Petersburg FL	55 Springfield MA-CT	78 Anchorage AK
12 Seattle WA	34 Louisville KY-IN	56 El Paso TX-NM	79 Sarasota-Bradenton FL
13 Washington DC-VA-MD	35 Nashville-Davidson TN	57 Wichita KS	80 Beaumont TX
14 Phoenix AZ	36 Tulsa OK	58 Little Rock AR	81 Eugene OR
15 Minneapolis-St. Paul MN	37 Memphis TN-MS-AR	59 Oxnard-Ventura CA	82 Pensacola FL-AL
16 St. Louis MO-IL	38 New Orleans LA	60 Charlotte NC-SC	83 Bakersfield CA
17 Denver-Aurora CO	39 Jacksonville FL	61 Knoxville TN	84 Lancaster-Palmdale CA
18 San Diego CA	40 Columbus OH	62 Honolulu HI	85 Indian-Cathdrl Cty-Plm Sprng CA
19 Pittsburgh PA	41 Salt Lake City UT	63 Las Vegas NV	86 Salem OR
20 Portland OR-WA	42 Birmingham AL	64 Akron OH	87 Cape Coral FL
21 San Jose CA	43 Tucson AZ	65 Charleston-No. Chrlston SC	88 Boulder CO
22 Kansas City MO-KS	44 Albuquerque NM figure	66 Fresno CA	89 Brownsville TX
23 Cleveland OH	45 Providence RI-MA	67 Grand Rapids MI	90 Laredo TX

surfaces, collect on buildings, painted and unpainted, (along with trees and other surfaces), and wash into the soil below (Mielke et al., 1984; Mielke, 1999; Rolfe and Haney 1975; US EPA, 1986). Lead in soils within non-smelter urbanized areas is derived from a mixture of Pb from paint and gasoline with a ratio that is spatially variable dependent on the proximity of roadways and/or age, condition, and maintenance of homes with exterior Pb paint (Wu et al., 2010). The characteristics of fine and ultra-fine particles of Pb collecting on building surfaces assists with understanding why, regardless of the building materials such as brick vs. painted siding, the soils next to buildings are generally more contaminated than soils away from buildings (Button, 2008; Clark et al., 2006; Linton et al., 1980; Mielke et al., 1984; Mielke, 1999; Schmitt et al., 1988).

Another comparison of Pb from paint and vehicle source was from a study that weighed the paint scraped from an old house and found that if the same house had been power-sanded, 7.4 kg of Pb would have been released as fine particles (Mielke et al., 2001). A substantial amount, considering that the total tolerable daily intake (TTDI) for a child under 6 years of age is 6 μg Pb from all sources (Ross et al., 2000). Converting 7.4 kg Pb to dust means an equivalent of more than

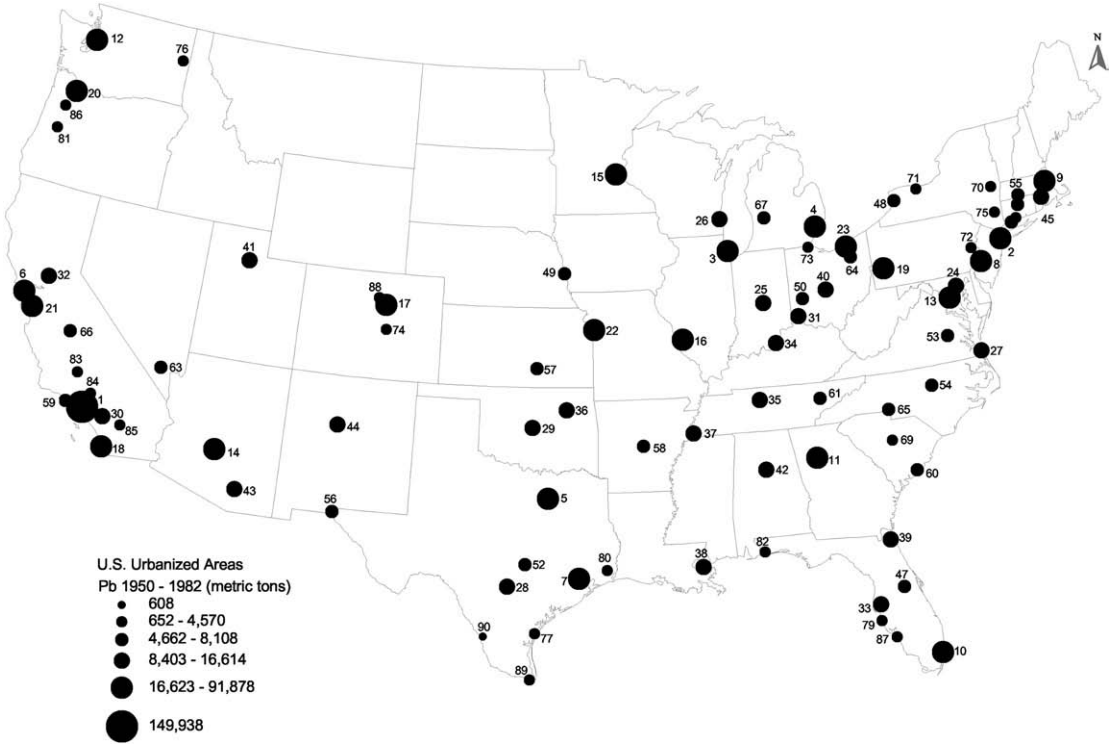

Fig. 2. Estimated lead aerosol inputs from gasoline into 90 US urbanized areas (UA) from 1950 to 1982. The numbers on the map are rankings of each UA. The size of each dot refers to the quartile for each group of UAs reported in Table 2, with the extremes, Los Angeles UA ranked 1 and Laredo, Texas ranked 90. Some of the UAs have been sites of soil Pb studies as indicated in Table 3.

1 billion (10^9) times the TTDI. For perspective with vehicle traffic related emission, the one-time release of 7.4×10^9 μg of Pb dust from power sanding compares with the *annual* emission of 50×10^9 μg of Pb dust per 0.1 mile (0.16 km) during the peak use of leaded gasoline from the nearby arterial street (Mielke et al., 2001).

4.2.2. Bioavailability of Pb particles

An important characteristic of anthropogenic Pb deposited in soil is that it speciated in easily absorbed carbonate, iron, and manganese hydroxide soil fractions, whereas natural Pb in soils consists of the non-bioavailable portion and is speciated in the residual fractions (Chlopecka et al., 1996; Lee et al., 1997). This means that soils contaminated by anthropogenic Pb aerosols from gasoline and paint are more bioavailable than the small portion of Pb associated with natural soils. This observation is supported by the ease of extraction of Pb from soils with a relatively mild 1 molar nitric acid at room temperature (Mielke et al., 1983, 2005a). This extraction gives similar results for Pb as boiling concentrated nitric acid extraction methods (US EPA, 1996b). In Los Angeles California, analysis of 550 soil samples indicated that total Pb (extracted using boiling concentrated nitric acid) and bioavailable Pb were highly correlated ($r=0.96$) (Wu et al., 2010).

The bioavailability and toxicology characteristics of Pb are related to particle size. The toxicity of Pb additives was indicated by the phenomenally rapid decline of blood Pb, especially for children, when the Pb additives were removed from gasoline (Annest et al., 1982). During the rapid phase-down beginning in January, 1986, the US childhood prevalence of blood Pb decreased by 77% (Pirkle et al., 1994), and similarly rapid declines of human blood Pb were experienced by every nation that eliminated Pb additives from gasoline (Thomas et al., 1999). Thus, gasoline Pb aerosols consisted of fine to ultra-fine particles which, after deposition, are easily extracted from soils, highly bioavailable and very toxic to humans, especially children.

4.3. US background and urban soil lead concentrations

Soils are the most accessible of the loose materials, or pedosphere, of the Earth. The United States Geological Survey undertook a survey of soil ($n=1323$) to establish the background metals in the US and found that the background soil Pb median is 16.5 mg/kg and ranges from 10.3 to 30.1 mg/kg (Gustavsson et al., 2001 p. 22). Even smaller quantities of Pb were identified in fresh alluvium from the Mississippi River, the parent materials of the urbanized area of New Orleans (Mielke et al., 2000). Thus, natural soils contain relatively small quantities of Pb. The practical meaning of this finding is that all UAs have an available source of low Pb soils nearby.

In contrast to background soil lead, significantly larger quantities of soil Pb are reported in urbanized areas. The remnants of Pb additives in gasoline are observed by studying the variations of Pb content of the pedosphere. Updating a review of urban soil Pb studies by Burgoon et al. (1995) assists with characterizing the material impact that Pb additives to gasoline may have had on environments and the health of inhabitants of urbanized areas. Table 3 is a list of soil Pb studies sorted by rank and group of urbanized areas according to the estimated quantity of Pb additives and Pb emissions, given in Tables 1 and 2, and illustrated in Fig. 2. Whenever possible, the number of samples, the median (or geometric mean), and the ranges are given in Table 3.

4.4. Characteristics of soil lead in urbanized areas

In Table 3, the highest density collections for urbanized areas to date have been conducted in Baltimore MD, cities of Minnesota, Milwaukee WI, cities of Louisiana (especially New Orleans), Illinois parks, Syracuse NY, and Detroit MI. One protocol developed in Minnesota for high density sampling involved stratifying samples by census tracts (enumeration districts) and collecting samples along residential streets, near foundations of residences, and from open spaces (Mielke, 1991, 1994; Schmitt et al., 1988). The protocol was revised to include busy streets in the second survey in metropolitan New Orleans, LA, where the median N of soil collection was 19 samples per km^2 (Mielke et al., 2005a). The following discussion lists six characteristics of UAs that were derived empirically from the high density sampling studies of urbanized areas.

4.4.1. Soil Pb, city size, inner-city vs. outer-city location

Larger UAs are locations where vehicle traffic input larger quantities of Pb aerosols than vehicle traffic input in smaller UAs. As a result, the storage of Pb in soils in larger cities is higher than storage of Pb in soils of smaller towns, a characteristic observed in Table 3 for Minnesota, Louisiana and Michigan. It is important to note that median refers to the middle Pb result of a given set of soil Pb results. The number (N) of samples collected for each of the cities ranged from 38 for Pontiac to 5467 for Survey 2 of New Orleans (see Table 3). In Table 3 the progression of median soil Pb (from larger to smaller cities) of Minneapolis, Saint Paul, Duluth, Saint Cloud and Rochester is a median of 230, 170, 144, 41 and 25 mg/kg Pb, respectively (Mielke et al., 1984/85, 1989, 1997; Mielke, 1993; Schmitt et al., 1988). Table 3 also shows that the median soil Pb for New Orleans, the largest Louisiana city, is in the >200–399 mg/kg range while the median soil Pb in the small community of Lafourche is <25 mg/kg (Mielke et al., 1997). The median soil Pb concentration of Metro New Orleans soil: Survey 1 = 167 mg/kg ($N=4026$), Survey 2 = 112 mg/kg ($N=5467$) (Mielke et al., 2005a). In Michigan, the median soil Pb for Detroit is 189 mg/kg while in the smaller city of Pontiac the median soil Pb is 86 mg/kg (Mielke et al., 2003).

Furthermore, soils of inner-city communities contain larger quantities of Pb than outlying communities of the same city. This was noted in a soil survey of urban gardens of Baltimore and then in Minneapolis-St. Paul (Mielke et al., 1983, 1984). LaBelle (1986) studied soil Pb in parks where buildings with Pb-based paint do not generally exist and noted that parks of inner-city Chicago, suburbs, and rural areas, have median soil Pb of 262, 87, and 37 mg/kg respectively. The soil in inner city of Milwaukee has a median soil Pb of 240 mg/kg while the suburban communities have a median Pb of 50 mg/kg (Brinkmann 1994b). The footprint of soil Pb in UAs requires taking into account both lead-based paints and nonpaint sources in order to comprehend the input of Pb in various sized cities and different locations within the same region (Mielke et al., 2001, 2008). LaBelle et al. (1987) studied soils that were not likely to be impacted by buildings or industrial sites and noted that Pb in Illinois soils vary widely depending on traffic density, distance from the busiest nearby roadway, and traffic volume. Laidlaw and Filippelli (2008) reviewed the quantity and distribution of soil Pb in numerous cities of North America, and to date, all urbanized areas exhibit the same city size and community location characteristics that have been described above.

4.4.2. Soil is an enormous Pb dust reservoir

Children characteristically expose themselves to Pb dust via hand-to-mouth activity (Sayre et al., 1974; Ko et al., 2007). Children's hands tested with wipes at childcare centers before and after outdoor play in New Orleans indicated that children had more Pb after playing outside than after playing inside (Viverette et al., 1996). If the children engaged in hand-to-mouth behavior at private inner-city childcare centers the amount of Pb per hand exceeded the 6 μg Total Tolerable Daily Intake (TTDI) of Pb by a factor of 5 or more; public ("Head Start") childcare centers, where play areas are covered with rubberized playground surfacing, children's hands did not exhibit Pb differences before and after outdoor play (Viverette et al., 1996). Another study by Nielsen and Kristiansen (2005) compared exposure to Pb on

Table 3
Soil lead studies in the US. Median Pb results in mg/kg. See Tables 1, 2 and Fig. 2.

		N	Min	Med	Max	Reference
	Background soil Pb, US	1319		16.5		Gustavsson et al. (2001)
Rank	*City-State*					
1-1st	Los Angeles, California	550	9		216,174	Wu et al. (2010)
1-1st	Los Angeles, California	343				Sutton et al. (1995)
1-1st	Chicago Illinois	57				Cannon and Horton (2008)
3-1st	Chicago Illinois	667				LaBelle et al. (1987)
3-1st	Chicago-Urban Parks	255	12	262	1312	LaBelle (1986)
3-1st	Chicago-Suburban Parks	245	12	87	1637	LaBelle (1986)
	Illinois, Rural Parks	177	12	37	937	LaBelle (1986)
4-1st	Detroit, Michigan	59	13	189	1345	Mielke et al., 2003, Supplement
4-1st	Detroit-Suburbs, Michigan	76	4	16	810	Mielke et al., 2003, Supplement
	Pontiac, Michigan	38	15	86	495	Mielke et al., 2003, Supplement
6-1st	Oakland, California	358	7		347,900	Sutton et al. (1995)
6-1st	Alameda, California	138	22		3187	Teichman et al. (1993)
9-1st	Boston, Massachusetts	195	7		13,240	Rabinowitz and Bellinger (1988)
10-1st	Miami, Florida	240	2		1060	Chirenje et al. (2004)
12-1st	Seattle, Washington	51	150		74,000	Roberts et al. (1991)
13-1st	Washington, D.C.	240	12		6015	Elhelu et al. (1995)
15-1st	Mpls/St Paul, Minnesota	90	5		7650	Mielke et al. (1984)
15-1st	Minneapolis, Minnesota	898	1	230	20,136	Schmitt et al. (1988)
15-1st	St. Paul, Minnesota	832	1	170	7994	Schmitt et al. (1988)
	Duluth, Minnesota	229	1	144	11,110	Schmitt et al. (1988)
	St. Cloud, Minnesota	124	1	41	1952	Schmitt et al. (1988)
	Rochester, Minnesota	165	1	25	1930	Schmitt et al. (1988)
	Outstate farms Minnesota	781	1	31	7111	Schmitt et al. (1988)
23-2nd	Cleveland, Ohio	50	19		811	Petersen et al. (2006)
24-2nd	Baltimore, Maryland	122	0.01		5620	Yesilonis et al. (2008)
24-2nd	Baltimore, Maryland	422	1	100	10,900	Mielke et al. (1983)
25-2nd	Indianapolis, Indiana	116	46		565	Filippelli et al. (2005)
26-2nd	Milwaukee, WI summary data	372	1	160	880	Brinkmann (1994b)
26-2nd	Milwaukee cent city + No and So Side	256	1	240	7220	Brinkmann (1994b)
26-2nd	Milwaukee, WI suburbs	122	1	50	1780	Brinkmann (1994b)
32-2nd	Cincinnati, Ohio	60	2		3166	Leeuwen et al. (1990)
31-2nd	Cincinnati, Ohio-Childcare centers	69	17		4636	Button (2008)
31-2nd	Cincinnati, Ohio	60				Tong (1990)
32-2nd	Sacramento, California	232	57	229	320,834	Sutton et al. (1995)
33-2nd	Tampa, Florida	146	<1	100	9160	Brinkmann (1994a)
38-2nd	New Orleans, LA Survey 1	4026	18	134	183,588	Mielke et al. (2005a)
38-2nd	New Orleans, LA Survey 2	5467	3	100	52,798	Mielke et al. (2005a)
38-2nd	LA Orleans Parish	~1540	<25	>200	<1000	Mielke et al. (1997)
	LA Lafourche Parish	~190		<25		Mielke et al. (1997)
44-2nd	Albuquerque, New Mexico	43	3		5280	Franz and Hadley (1981)
49-3 rd	Omaha, Nebraska	176				Angle and McIntire (1979)
50-3 rd	Dayton, Ohio	22	22		461	Ritter and Rinefierd (1983)
56-3 rd	El Paso, Texas	94	<20		8700	Pingitore et al. (2009)
62-3 rd	Honolulu, HI	18				Fu et al. (1988)
65-3 rd	Charleston So. Carolina	164	9		7890	Galke et al. (1975)
68-4th	New Haven, Connecticut	487	30		7000	Stark et al. (1982)
77-4th	Corpus Christi, Texas	485	21		2969	Harrison (1987)
	Pueblo, Colorado	33				Diawara et al. (2006)
	Connecticut	174	<10		2200	Stilwell et al. (2008)
	Gainesville, Florida	202	2.13		1091	Chirenje et al. (2004)
	Champaign Illinois	116	20		1061	Solomon and Hartford (1976)
	Louisiana and Minnesota	6,342	Urban–rural comparisons.			Mielke (1993)
	S.E. Michigan	171	3		7400	Murray et al. (2004)
	Mt. Pleasant, Michigan	189	100		16,839	Francek (1992)
	Syracuse, New York	2998	45			Griffith et al. (2009)
	Syracuse, New York	162				Griffith (2002)
	Syracuse, New York	194		80 (GM)		Johnson and Bretsch (2002)
	Lubbock, Texas	52		35		Brown et al. (2008)
	Maine urban soils	100				Kruger and Duguay (1989)

children's hands at three kindergartens before and after remediation. One kindergarten had very small amounts of Pb in soil (control) and two kindergartens had soil Pb concentrations of 100–200 mg/kg. Following remediation of the 100–200 soil Pb to below 10 mg/kg the study found a good agreement (with some overlap) between the average concentration of Pb in soil and the amount of Pb on the hands of the children (Nielsen and Kristiansen, 2005).

One critical characteristic is Pb loading of the soil surface. For example, in a study in New Orleans measuring the Pb loading of the surface of bare soil it was noted that a soil containing 400 mg/kg (the current EPA standard for soil Pb) exceeds the interior floor guideline of Pb loading (currently 40 μg/ft^2) by a factor of about 35 (Mielke et al., 2007a). Children are also exposed to soil Pb by track-in of contaminated soil into homes on shoes (Hunt et al., 2006) family pets,

and resuspension and deposition of Pb contaminated urban soil dust which penetrates interiors of homes and settles onto contact surfaces (Laidlaw and Filippelli, 2008; Layton and Beamer, 2009; Paustenbach et al., 1997; Polidori et al., 2009). Considering the quantity of Pb loading on the soil surface, it is easy to understand how Pb contaminated exterior soil results in children picking up Pb on their hands as well as the large potential for track-in of exterior Pb into building interiors.

4.4.3. Association of soil Pb and blood Pb

Soil Pb concentrations have been associated with children's blood Pb concentrations using multiple study designs—cross-sectional, ecological spatial, ecological temporal, prospective soil removal, and isotopic studies (Laidlaw and Filippelli, 2008). Comparing various communities, both soil Pb and blood Pb of children living in smaller towns' exhibit lower blood Pb than children living in larger cities (Mielke et al., 1989, 1997; Mielke and Reagan 1998). To observe this characteristic requires full spectrum blood Pb surveillance rather than truncated blood Pb at an arbitrary level such as 10 or 15 μg/dL. In New Orleans the blood Pb response of children to soil Pb is curvilinear (Mielke et al., 1997, 1999, 2007b). Johnson and Bretsch (2002) observed a similar curvilinear relationship between soil Pb and children's blood Pb in Syracuse, NY. The most recent New Orleans urban soil Pb and blood Pb study shows the following results: below 100 mg/kg soil Pb children's blood Pb response is steep at 1.4 μg/dL per 100 mg/kg, between 100 mg/kg and 300 mg/kg the curve flexes, and above 300 mg/kg soil Pb children's blood Pb response becomes gradual 0.32 μg/dL per 100 mg/kg (Mielke et al., 2007b). Soil Pb levels are also associated with a risk of blood Pb concentrations greater than 10 μg/dL at soil Pb concentrations lower than indicated by soil Pb–blood Pb curves (Mielke et al., 2007b). For example, Malcoe et al. (2002) found that logistic regression of yard soil Pb >165.3 mg/kg was associated independently with blood Pb greater than or equal to 10 μg/dL (OR, 4.1; CI, 1.3–12.4). Similarly, the Texas Department of Health (2004) used a large database from El Paso, Texas Area and found an odds ratio 4.5 (1.4, 14.2) for the relationship between a 500 mg/kg increase in soil Pb above the background and blood Pb level >10 μg/dL. Similar soil Pb and blood Pb responses of children are expected as a general characteristic in other UAs of the US. The close connection between children and their environment is one of the critical qualities shared as part of the developmental processes of children (Louis et al., 2006).

4.4.4. Blood Pb seasonality and Pb dust resuspension

Lead dust is so ubiquitous, especially within the 1st and 2nd group of urbanized areas (Tables 1 and 2), that seasonal wet–dry weather patterns characteristically influence the blood Pb of children. This characteristic was described by Laidlaw et al. (2005) for three cities with climates as different as Syracuse, NY, Indianapolis, IN, and New Orleans, LA. In all three cities blood Pb increased during droughty periods when soil is dusty and decreased blood Pb during rainy periods when soil dust is settled (Laidlaw et al., 2005). Similarly, in Mexico City, Rosas et al. (1995) observed that during rainy seasons of the year, PM_{10} dust was settled and atmospheric Pb concentrations were lower; during seasons with low rainfall PM_{10} and atmospheric Pb concentrations increased. In Milwaukee, Wisconsin, blood Pb levels also follow a seasonal pattern with peaks in the summer and autumn (Havlena et al. 2009). They observed that particulate matter less than $PM_{2.5}$ correlated with the seasonal variation in 10 month old children's blood Pb, and suggested that the Pb in the $PM_{2.5}$ was causally related to seasonal variations in children's blood Pb levels.

The characteristic of Pb contaminated soil being resuspended is supported by Pingitore et al. (2009) who used synchrotron-based XAFS (X-ray absorption fine structure) to quantify the Pb species in the air of El Paso. Pingitore et al. (2009) concluded that the soil is the dominant source of Pb, and that it was being resuspended as aerosols into the atmosphere. Harris and Davidson (2005) calculated that annually at least 54 MT of Pb aerosol particles in the Southern California Air Basin were being resuspended from soils contaminated by previous Pb additives in gasoline. Climates differ markedly between the UAs shown in Fig. 2, and children's blood Pb responses to soil Pb are expected to be related to local weather factors that affect resuspension in each UA.

Pb dust in urban environments can result in elevated lead loading of both interior and exterior contact surfaces (Caravanos, et al., 2006a, b). Lead loading (mass/unit area) is well known to correlate with urban children's blood lead levels. However, exterior lead loading is not routinely measured in US cities, and is likely a better measure of risk to children blood lead levels than air lead concentrations (mass/volume). Caravanos et al. (2006b) demonstrated that exterior lead loading in the five boroughs of New York City was highly elevated when compared to the HUD/EPA indoor Pb in dust standard of 40 μg/ft^2. Caravanos et al. (2006b) measured the following median dust loadings in New York: Brooklyn (730 μg/ft^2), Staten Island (452 μg/ft^2), the Bronx (382 μg/ft^2), Queens (198 μg/ft^2) and Manhattan (175 μg/ft^2). In a related study, Caravanos et al. (2006a) demonstrated how exterior particulate Pb can accumulate rapidly on interior surfaces. They observed that interior settled dust in a Pb-free room with a window slightly open exceeded the HUD/EPA indoor Pb in dust standard of 40 μg/ft^2 (431 μg/m^2) within a 6 week period.

4.4.5. Association between blood lead <10 μg/dL and children's clinical health outcomes

Low blood lead levels are also associated with a reduction in children's intelligence. Canfield et al. (2003) observed that when lifetime average blood lead concentrations in children increased from 1 to 10 μg/dL, the intelligence quotient (IQ) declined by 7.4 points. Jusko et al. (2008) observed that compared with children who had lifetime average blood lead concentrations <5 μg/dL, children with lifetime average concentrations between 5 and 9.9 μg/dL scored 4.9 points lower on Full-Scale IQ (91.3 vs. 86.4, $p=0.03$). Similarly, Surkan et al. (2007) observed that children with 5–10 μg/dL had 5.0 (S.D. 2.3) points lower IQ scores compared to children with blood lead levels of 1–2 μg/dL ($p=0.03$). Interestingly, multiple studies have shown that the strongest lead effects on IQ occurred within the first few micrograms of blood Pb (Schnaas et al., 2006; Canfield et al., 2003; Lanphear et al., 2005). Low blood lead levels have also been associated with various physiological outcomes such as kidney damage (Fadrowski et al., 2010), puberty delay in boys (Williams et al., 2010) and girls (Selevan et al., 2003) and cardiovascular outcomes in adults (Navas-Acien et al., 2007).

4.4.6. Soil Pb, blood Pb and school achievement

Lower blood Pb levels (<10 μg/dL) typically associated with urban soil Pb exposure are now being associated with health outcomes related to subtle nervous disorders. Needleman et al. (1979) identified the impact of children's early Pb exposure to classroom performance. Low blood Pb <10 μg/dL is associated with ADHD (Nigg et al., 2010) and a reduction in children's test scores for reading (OR 0.51, $p=0.006$) and writing (OR 0.49, $p=0.003$) and mathematics (Chandramouli et al., 2009; Miranda et al., 2007). Reduced student performance has been demonstrated as one of the characteristics of the accumulation of Pb dust in communities of UAs. The literature of the effects of Pb on student performance is large (Martin, 2008).

Support for the idea that soil Pb in a community is associated with student performance was indicated in Minnesota by the observation that dropout rates of high school students followed city size and soil Pb (Mielke et al., 1989). In New Orleans, soil Pb on the play areas of elementary public schools follow the same trend as the urban soil Pb map of the entire city; however, soils on school grounds are significantly less Pb contaminated than soils of neighboring residential properties where the most vulnerable preschool children reside

and play before they attend school (Higgs et al., 1999). New Orleans school achievement test scores were found to be associated with soil metals including Pb (Mielke et al., 2005b; Mielke and Berry, 2007, pp 116–123). Finally, standardized test performance of children of local schools is significantly linked with soil Pb and blood Pb of the same school district and this phenomenon indicates the serious neurotoxicity effects of Pb (Zahran et al., 2009). These studies characterize the more insidious disparities and costly influences that environmental Pb imposes on urban society (Campanella and Mielke, 2008; Chandramouli et al., 2009; Gould, 2009; Muennig, 2009; Nigg et al., 2008; Zahran et al., 2009).

4.5. Precautionary approaches for primary exposure prevention

As described in the sections above, the current consensus is that for children a blood Pb of 5 μg/dL or even 2 μg/dL will cause clinical signs. The critical problem for children Pb is to successfully curb the blood Pb in the 2–10 μg/dL range which, as reported by Gould (2009), currently prevails among 24.5%, or 9.6 million US children. To reduce the Pb exposure to meet lower guidelines, large US urbanized areas will probably require extensive environmental treatment, and soil Pb is one factor that can be changed to reduce children's Pb burdens (Filippelli and Laidlaw, 2010). Fortunately, as described previously clean soil with a median Pb content of 16.5 mg/kg is available nearby all US urbanized areas (Gustavsson et al., 2001).

In Minneapolis a pilot project was conducted that successfully reduced children's exposure and prevented the expected seasonal summertime blood Pb increases (Mielke et al., 1992). The method was refined in New Orleans where abundant and remarkably clean (median ~5 mgPb/kg) Mississippi River sediments are available as a natural resource for covering Pb contaminated urban soils (Mielke, 2005; Mielke et al., 2006a,b). Other pilot projects focused on soil Pb were conducted in Boston and Chicago with varying success (US EPA, 2001; Binns et al., 2004).

A full-scale national program is underway in Norway to clean up contaminated soils at all childcare centers, elementary schools and parks in the ten largest cities (Ottesen et al., 2008). The differences between the US approach and Norway's program are striking: in the US secondary prevention is conducted after a child is identified with elevated blood Pb (CDC blood Pb guideline is ≥10 μg/dL, or even higher depending on local health department jurisdictions). In Norway, the WHO principles of the precautionary approach are followed (Louis et al., 2006). Therefore, the emphasis is on primary prevention whereby environmental contaminants are directly addressed and treated; children's blood Pb samples are not included as part of the protocol. In the US the current residential soil guideline is 400 mg/kg Pb for the bare soil of a play area, and 1200 mg/kg for the remaining areas of the property. In Norway the soil Pb guideline for play areas is 100 mg/kg, and there is discussion about reducing the guideline to 60 mg/kg.

Research in New Orleans indicates that communities with a median soil Pb ≤80 mg/kg would generally prevent children from reaching a blood Pb ≥10 μg/dL (Mielke et al., 1999). If a stricter blood Pb guideline is observed that reflects the current clinical health effects of Pb, then the soil Pb guideline must also be significantly lower. As shown in Table 3, many US urbanized areas have soil Pb medians above 100 mg/kg. Norway's precedence for implementing a national clean play area program serves as a model for commitment to primary prevention that benefits children and ultimately all community members of Pb impacted urbanized areas (Ottesen et al., 2008).

5. Conclusions

The principal message from the Pb emission estimates for 90 urbanized areas is that precaution must accompany the selection of fuel additives used in urban-industrial society. Lead additives in fuel created the situation whereby vehicles traveling on roadways functioned as a Pb dust delivery system into the most populated communities of urbanized areas. As a result, the commercial use of Pb additives in gasoline had the consequence of materially contaminating urbanized areas, with 100's to over 100,000 MT of Pb dust, depending directly on vehicle traffic patterns in urbanized areas. Although Pb additives were removed from vehicle fuels for highway use, the mass of Pb dust inputs continue to materially impact the environment. Lead dust is associated with an array of chronic health problems of the people living in urbanized areas.

Lead-based paint coating is visible and presents a hazard when it deteriorates or is treated in a manner that creates Pb dust. Past use of Pb additives in gasoline resulted in ~75% emitted as tiny Pb aerosols particles that were essentially invisible. Empirical means for visualizing the material impact of both paint and nonpaint sources of Pb on urbanized areas involves soil Pb mapping. High density soil Pb mapping (i.e., ~19 samples per km^2) has been conducted in a few cities, but as indicated in Table 3, most urban soil Pb surveys have insufficient sample density to guide intervention efforts. All US cities are in close proximity to soils containing 10.3–30.1 mg/kg Pb thereby making intervention with clean soils possible for every city.

Young children have no possibility of creating safe environments for themselves—this is an adult responsibility. Characterization of Pb additives in gasoline and its legacy of effects on urbanized areas should result in better understanding of actions needed for hazard control to protect communities from the health effects of both paint and nonpaint sources of Pb.

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CHAPTER 4

Soil Pb and children's blood Pb levels are associated spatially and temporally in urban areas: A new paradigm pointing towards a cost-effective solution.

This chapter contains a collection of four papers showing how atmospheric soil and atmospheric Pb correlate in Birmingham, Chicago, Detroit and Pittburgh (USA), atmospheric soil correlates spatially with children's PbB levels in New Orleans, Louisiana (USA), and atmospheric Pb and atmospheric soil correlate temporally with children's PbB levels in Detroit, Michigan (USA). We also argue that this research regarding the causes of swings in seasonal PbB levels present a new paradigm of the exposure pathway of children to Pb and point to a relatively simple and cost-effective way toward reducing the Pb load for urban youth (Filippelli and Laidlaw, 2010).

Paper 1: "*Re-suspension of Pb contaminated urban soil as a dominant source of atmospheric Pb in Birmingham, Chicago, Detroit and Pittsburgh, USA."*

MAS Laidlaw, S Zahran, HW Mielke, MP Taylor, GM Filippelli

Published in *Atmospheric Environment*.

In order to explore the hypothesis that airborne urban soil contributes to the burden of Pb aerosols, one of the objectives of the *Atmospheric Environment* (Laidlaw et al., 2012) study was to analyse temporal variations in atmospheric soil and Pb aerosols in four US cities: Pittsburgh, Detroit, Chicago, and Birmingham. The specific goals of the study were to test whether re-suspended urban soil was the dominant source of Pb aerosols in the four cities, and whether atmospheric soil and Pb aerosols follow seasonal patterns and if the highest concentrations occurred during the summer and/or autumn. Additionally, the study examines

whether atmospheric soil and Pb aerosol concentrations were lower on the weekends and Federal Government holidays relative to weekdays to evaluate the possibility that automotive turbulence causes re-suspension of Pb contaminated urban soil to produce increases in Pb aerosols into the urban atmosphere.

Author Contributions (Laidlaw et al., 2012):

MAS Laidlaw: 60%

Concept, data acquisition, statistics, writing, editing

S. Zahran: 25%

Statistics, writing, editing.

HW Mielke: 5%

Editing

MP Taylor: 5%

Mentoring, editing**.**

GM Filippelli: 5%.

Editing.

Paper 2: "*Linking Source and Effect: Re-suspended Soil Pb, Air Pb, and Children's PbB Levels in Detroit, Michigan"*

Zahran S., **MAS Laidlaw**, S McElmurry, MP Taylor, GM Filippelli

Published in *Environmental Science and Technology* (2013)

As compared to the reference month of January, child blood lead levels (BLLs) in Detroit are found to be between 11% and 14% higher in the months of July, August, and September. Explaining this seasonal phenomenon is the aim of our Environmental Science and Technology paper (Zahran et al 2013a), and it is our contention that any theory of contemporary Pb risk must logically account for this striking empirical observation.

Author Contributions (Zahran et al., 2013a):

S Zahran: 40%

Statistical analysis, writing, editing.

MAS Laidlaw: 30%

Concept, study design, writing, editing.

S McElmurry: 20%

Writing, editing, submission.

MP Taylor: 5%

Editing, mentorship.

GM Filippelli: 5%

Editing.

Paper 3: "*Advancing upstream prevention in New Orleans, Louisiana, USA. Determining the relative importance of soil sample locations to predict risk of child Pb exposure."*

S Zahran, HW Mielke, GM Filippelli, SP McElmurry, **MAS Laidlaw**, MP Taylor

Published in *Environment International*.

A major purpose of the second *Environment International* (Zahran et al., 2013b) study is to evaluate the soil sample location(s) which best explain the spatial variation in children's PbB concentrations, in order to minimize soil sampling in any future soil Pb mapping exercises. This involved examining the variance explained by a regression model with children's PbB as a dependent variable and soil samples collected from four soil sample location types as independent variables: house-side, residential street-side, busy street-side and open space.

Author Contributions (Zahran et al., 2013b):

S Zahran: 50%

Statistical analysis, writing, editing.

HW Mielke: 20%

Statistical analysis, writing, editing.

GM Filippelli: 7.5%

Editing, writing.

SP McElmurry: 7.5%

Editing, writing.

MAS Laidlaw: 7.5%

Editing, writing.

MP Taylor: 7.5%

Editing, writing.

Paper 4: "*The Elephant in the Playground: Confronting Pb-contaminated soils as an important source of Pb burdens to urban populations*."

GM Filippelli, **MAS Laidlaw**.

Published in *Perspectives in Biology and Medicine*.

In this paper, we argue that our recent research regarding the causes of swings in seasonal PbB levels present a new paradigm of the exposure pathway of children to Pb and point to a relatively simple and cost-effective way toward reducing the Pb load for urban youth (Filippelli and Laidlaw, 2010).

Author Contributions (Filippelli and Laidlaw, 2010):

GM Filippelli: 70%

Concept, writing, submission.

MAS Laidlaw: 30%

Original PbB seasonality concept, writing, editing.

Chapter 4 - Paper 1: "*Re-suspension of Pb contaminated urban soil as a dominant source of atmospheric Pb in Birmingham, Chicago, Detroit and Pittsburgh, USA.*"

Authors: MAS Laidlaw, S Zahran, HW Mielke, MP Taylor, GM Filippelli

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Re-suspension of lead contaminated urban soil as a dominant source of atmospheric lead in Birmingham, Chicago, Detroit and Pittsburgh, USA

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ABSTRACT

Soils in older areas of cities are highly contaminated by lead, due largely to past use of lead additives in gasoline, the use of lead in exterior paints, and industrial lead sources. Soils are not passive repositories and periodic re-suspension of fine lead contaminated soil dust particulates (or aerosols) may create seasonal variations of lead exposure for urban dwellers. Atmospheric soil and lead aerosol data from the Interagency Monitoring of Protected Visual Environments (IMPROVE) database were obtained for Pittsburgh (Pennsylvania), Detroit (Michigan), Chicago (Illinois), and Birmingham (Alabama), USA. In this study the temporal variations of atmospheric soil and lead aerosols in these four US cities were examined to determine whether re-suspended lead contaminated urban soil was the dominant source of atmospheric lead. Soil and lead-in-air concentrations were examined to ascertain whether lead aerosols follow seasonal patterns with highest concentrations during the summer and/or autumn. In addition, atmospheric soil and lead aerosol concentrations on weekends and Federal Government holidays were compared to weekdays to evaluate the possibility that automotive turbulence results in re-suspension of lead contaminated urban soil. The results show that the natural logs of atmospheric soil and lead aerosols were associated in Pittsburgh from April 2004 to July 2005 ($R^2 = 0.31$, $p < 0.01$), Detroit from November 2003 to July 2005 ($R^2 = 0.49$, $p < 0.01$), Chicago from November 2003 to August 2005 ($R^2 = 0.32$, $p < 0.01$), and Birmingham from May 2004 to December 2006 ($R^2 = 0.47$, $p < 0.01$). Atmospheric soil and lead aerosols followed seasonal patterns with highest concentrations during the summer and/or autumn. Atmospheric soil and lead aerosols are 3.15 and 3.12 times higher, respectively, during weekdays than weekends and Federal Government holidays, suggesting that automotive traffic turbulence plays a significant role in re-suspension of contaminated roadside soils and dusts. In order to decrease urban lead aerosol concentrations, lead deposition and subsequent children's seasonal exposure, lead contaminated urban soils need remediation or isolation because the legacy of lead continues to pose unnecessary and preventable health risks to urban dwellers.

1. Introduction

1.1. Lead dust contaminated urban soils

During the 20th century massive quantities of lead (Pb) were used in commercial and industrial products which contaminated soil. In the 1970s, the presumed dominant source of soil Pb contamination was Pb-based house paint (Ter Haar and Aronow, 1974). A subsequent study of garden soils conducted in metropolitan Baltimore, Maryland, raised questions about that assumption; soil around Baltimore's inner city buildings, predominantly unpainted brick, exhibited the highest amounts of Pb, while the

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soils outside of the inner city, where buildings were commonly constructed with wood siding coated with Pb-based paint, contained comparatively low amounts of Pb (Mielke et al., 1983). These findings suggested that Pb-based house paint could not fully account for the observed patterns of urban soil Pb and that a different source and process of contamination needed to be considered.

The mass of Pb additives used in gasoline in the US has been documented in detail by Mielke et al. (2010). In the US, motor vehicles used gasoline containing tetraethyl Pb additives from the 1920s to 1995. By the 1950s, Pb additives were contained in virtually all grades of gasoline. By 1986, when leaded gasoline underwent a rapid phase-down but not yet completely withdrawn, 5–6 million metric tons of Pb had been used as a gasoline additive across the US, of which, about 75% was released into the atmosphere (Chaney and Mielke, 1986; Mielke and Reagan, 1998). Thus, an estimated 4–5 million tons of Pb has been deposited into the US environment by way of gasoline-fueled motor vehicles (Mielke, 1994; Mielke et al., 2011). Finally, the accumulation of soil Pb from leaded gasoline emissions has been shown to be proportional to highway traffic volume (Mielke et al., 1997).

1.2. Soil re-suspension

Urban soil re-suspension has been studied by using the Interagency Monitoring of Protected Visual Environments database (IMPROVE, 2011). Laidlaw and Filippelli (2008) demonstrated that soils at eight sites located across America become re-suspended into the atmosphere during summertime and autumn when soils are dry and evapotranspiration maximized. Wells et al. (2007) documented the same phenomena at 15 IMPROVE locations in the western United States. Similarly, in Bakersfield, California, Young et al. (2002) observed that 74% of PM10 from July through September 1988 was composed of soil.

1.3. Re-suspension of urban soil as a source of lead aerosols

Limited data has been published on the seasonal variations in atmospheric Pb in the United States. However, summertime peaks of atmospheric Pb have been observed in Washington D.C. (Green and Morris, 2006; Melaku et al., 2008), Boston (USEPA, 1995), New York (Billick et al., 1979) New Jersey (Edwards et al., 1998; Yiin et al., 2000), and Chicago (Paode et al., 1998). Therefore, in order to explore the hypothesis that airborne urban soil contributes to the burden of Pb aerosols, one of the objectives of this study was to analyze temporal variations in atmospheric soil and Pb aerosols in four US cities: Pittsburgh, Detroit, Chicago, and Birmingham (Fig. 1). The specific goals of the study were to test whether re-suspended urban soil was the dominant source of Pb aerosols in the four cities, and whether atmospheric soil and Pb aerosols follow seasonal patterns and if the highest concentrations occurred during the summer and/or autumn. Additionally, the study examines whether atmospheric soil and Pb aerosol concentrations were lower on the weekends and Federal Government holidays relative to weekdays to evaluate the possibility that automotive turbulence causes re-suspension of Pb contaminated urban soil to produce increases in Pb aerosols into the urban atmosphere. Finally, we discuss the possible link between re-suspension of Pb contaminated urban soil as a contributor to a substantial public health issue, especially for children (Filippelli et al., 2005; Jones et al., 2009; Lanphear et al., 2002).

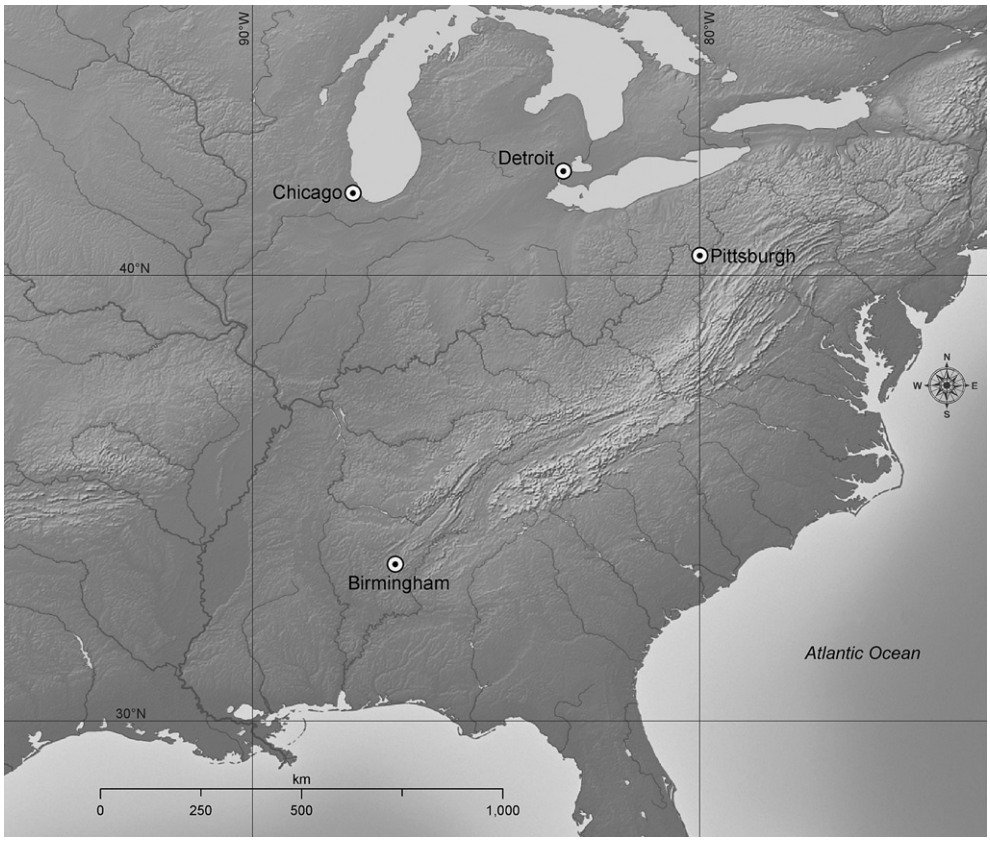

Fig. 1. The locations of Pittsburgh (Pennsylvania), Detroit (Michigan), Chicago (Illinois), and Birmingham (Alabama) in the USA, the cities examined in this study.

Table 1
Descriptive statistics of variables analyzed.

	Pittsburgh		Detroit		Chicago		Birmingham		Overall	
	N	Mean (Std. Dev)	*N*	Mean (Std. Dev)	*N*	Mean (Std. Dev)	*N*	Mean (Std. Dev)	*N*	Mean (Std. Dev)
Humidity	147	74.51 (13.95)	192	70.34 (12.58)	213	65.23 (13.16)	309	69.14 (12.92)	861	69.35 (13.40)
Sea level pressure (mb)	147	2,071 (13.94)	192	2,072 (15.48)	213	2,071 (14.25)	309	2,073 (10.94)	861	2,072 (13.41)
Temperature (°C)	147	12.39 (9.39)	192	8.56 (10.74)	213	10.38 (10.91)	309	18.46 (7.93)	861	13.22 (10.46)
Visibility (km)	147	13.26 (3.30)	192	14.10 (2.79)	213	14.42 (2.56)	309	14.05 (2.48)	861	14.02 (2.74)
Wind (km h^{-1})	147	11.61 (4.78)	192	12.77 (5.32)	213	15.14 (5.74)	309	9.15 (5.19)	861	11.86 (5.78)
Weekends/holidays	147	0.31 (0.47)	193	0.33 (0.47)	213	0.32 (0.47)	309	0.32 (0.47)	862	0.32 (0.47)
Air soil	146	0.99 (0.66)	193	0.812 (0.54)	213	0.810 (0.51)	309	1.964 (1.806)	861	1.255 (1.287)
Air lead	147	0.013 (0.014)	193	0.005 (0.003)	213	0.006 (0.004)	309	0.223 (0.033)	862	0.013 (0.022)

Table 2
Ordinary least squares and random effects generalized least squares regression models predicting the natural log of air Pb as a function of the natural log of air soil.

	Pittsburgh marginal effect	Detroit marginal effect	Chicago marginal effect	Birmingham marginal effect	Overall marginal effect
Air soil	0.709*** (0.088)	0.848*** (0.063)	0.710*** (0.070)	0.922*** (0.056)	0.841*** (0.034)
Constant	−4.574*** (0.059)	−5.305*** (0.040)	−5.104*** (0.046)	−4.713*** (0.049)	−4.898*** (0.174)
N	146	193	213	309	861
F	64.92	180.84	104.58	269.69	
$R^{2\ \text{overall}}$	0.3108	0.4863	0.3214	0.4677	0.5046
$R^{2\ \text{within}}$					0.4185
$R^{2\ \text{between}}$					0.7548
Wald X^2					621.13
N cities					4

Note: Standard errors in parentheses. ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$.

2. Methods

2.1. Data

Mielke et al. (2011) ranked the top 90 cities in the United States by the amount of Pb additives emitted from automobiles during 1950–1982. These included Pittsburgh, Detroit, Chicago, and Birmingham, which were ranked 19th, 4th, 3rd and 42nd, respectively. Atmospheric soil and Pb aerosol data were obtained from the Interagency Monitoring of Protected Visual Environments (IMPROVE) stations in each of the above named cities (IMPROVE, 2011). Atmospheric soil and Pb aerosol data were obtained for the following time periods: Pittsburgh, April 2004–July 2005; Detroit, November 2003–July 2005; Chicago, November 2003–August 2005; and Birmingham, May 2004–December 2006. Air monitoring station data were provided at the daily time interval. The four cities represent a range sizes, climatic regions, and atmospheric Pb aerosol levels.

To derive atmospheric soil estimates, IMPROVE provides a mineral equation of the assumed primary constituents in soil, including Al, Si, Ca, Fe, and Ti (IMPROVE soil estimation calculation, 2003). Soil composition variability is derived by the quadratic sum of constituent concentrations, assuming independence of measurement uncertainties:

$$[\mathrm{d(soil)}]^2 = [2.20 \times \mathrm{d(Al)}]^2 + [2.49 \times \mathrm{d(Si)}]^2 + [1.63 \times \mathrm{d(Ca)}]^2 + [2.42 \times \mathrm{d(Fe)}]^2 + [1.94 \times \mathrm{d(Ti)}]^2.$$

In addition to atmospheric soil and Pb aerosol data, we gathered local weather data on variables known to influence seasonality of atmospheric concentrations of soil and Pb, including average relative humidity (%), average sea level pressure (mb), average temperature (°C), average visibility (km), and average wind speed (km h^{-1}) (cf. Laidlaw and Filippelli, 2008). Descriptive statistics on atmospheric soil and Pb aerosol conditions as well as weather conditions in Pittsburgh, Detroit, Chicago, and Birmingham are

Table 3
Random effects generalized least squares regression coefficients predicting Air Pb and Air soil.

	Air lead Model 1	Air lead Model 2	Air soil Model 3	Air soil Model 4
	b	beta	*b*	beta
Humidity	−0.006* (0.003)	−0.073* (0.038)	−0.009*** (0.002)	−0.126*** (0.024)
Sea level pressure	0.008*** (0.177)	0.103*** (0.034)	0.007*** (0.002)	0.100*** (0.022)
Temperature	0.028*** (0.003)	0.288*** (0.033)	0.038*** (0.002)	0.400*** (0.021)
Visibility	−0.049*** (0.014)	−0.135*** (0.037)	−0.031*** (0.009)	−0.084*** (0.024)
Wind speed	−0.068*** (0.005)	−0.393*** (0.033)	−0.034*** (0.004)	−0.198*** (0.021)
Weekend/holiday	−0.122** (0.0600)	−0.122** (0.060)	−0.289*** (0.038)	−0.289*** (0.038)
Constant	−19.327*** (5.358)	−4.910*** (0.034)	−14.45*** (3.44)	0.024 (0.022)
N	861		860	
$R^{2\ \text{within}}$	0.2159		0.4025	
$R^{2\ \text{between}}$	0.8407		0.9867	
$R^{2\ \text{overall}}$	0.3362		0.5034	
Wald X^2	432.49		864.65	
N cities	4		4	

Note: Standard errors in parentheses. ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$.

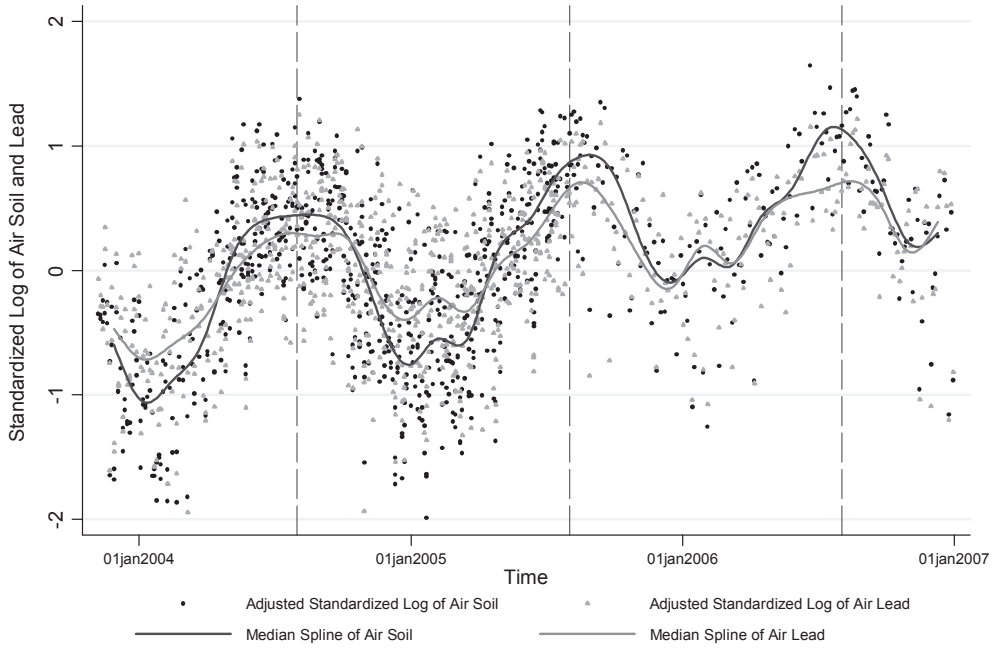

Fig. 2. Weather adjusted air Pb and air soil over time, including median spline fits, for Pittsburgh, Detroit, Chicago and Birmingham.

presented in Table 1. Data show that average atmospheric soil (1.96 $\mu g\,m^{-3}$) and Pb (0.22 $\mu g\,m^{-3}$) conditions are highest in Birmingham, followed by Pittsburgh (0.99 $\mu g\,m^{-3}$, 0.013 $\mu g\,m^{-3}$), Chicago (0.81 $\mu g\,m^{-3}$, 0.006 $\mu g\,m^{-3}$), and Detroit (0.81 $\mu g\,m^{-3}$, 0.005 $\mu g\,m^{-3}$).

2.2. Statistical procedures

To address the correlation between atmospheric Pb and soil, we used a *random effects generalized least squares model. The random effects model* allows each city to have its own intercept, yielding a weighted average of *between* and *within* city effects. Letting j denote the city, i denote the day an atmospheric Pb reading is taken, and y_{ij} denoting the atmospheric Pb level on day i in city j. Our regression estimator is modeled as: $y_{ij} = \beta_0 + \beta_1 S_{ij} + u_j + e_{ij}$, where, β_0 is the average air Pb across cities, and S_{ij} is the daily observation of atmospheric soil in a given city. The model divides the residual term into two components: (i) a city-specific error component, given by u_j; and (ii) a station specific error component, which varies between daily air station reading and city, given by e_{ij}. The city level residual u_j is the difference between city j's atmospheric Pb mean and the overall mean, with the mean of

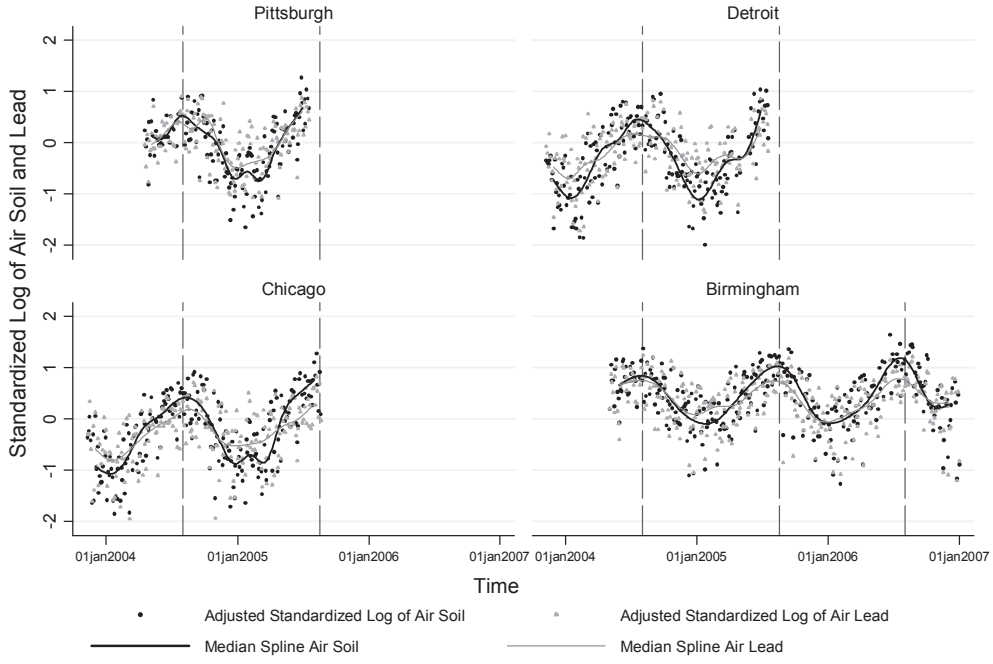

Fig. 3. Weather adjusted air Pb and air soil over time, including median spline fits, for Pittsburgh, Detroit, Chicago and Birmingham.

Table 4
Ordinary least squares and random effects generalized least squares regression coefficients predicting Air Pb and Soil.

	Pittsburgh		Detroit		Chicago		Birmingham		Overall	
	AL	AS	AL	AS	AL	AS	AL	AS	AL	AS
February	0.072 (0.178)	0.050 (0.179)	0.200 (0.141)	0.252* (0.138)	0.223 (0.151)	0.260* (0.145)	0.098 (0.135)	0.053 (0.134)	0.164* (0.085)	0.178** (0.088)
March	−0.041 (0.173)	−0.064 (0.174)	0.340*** (0.133)	0.477*** (0.131)	0.067 (0.145)	0.303** (0.139)	0.155 (0.133)	0.265** (0.132)	0.146* (0.082)	0.279*** (0.085)
April	0.165 (0.157)	0.653*** (0.158)	0.423*** (0.141)	0.865*** (0.138)	0.326** (0.147)	0.822*** (0.141)	0.215 (0.131)	0.409*** (0.130)	0.305*** (0.082)	0.707***(0.085)
May	0.391*** (0.150)	0.791*** (0.151)	0.612*** (0.149)	1.12*** (0.146)	0.565*** (0.149)	1.03*** (0.143)	0.418*** (0.121)	0.634*** (0.120)	0.563*** (0.079)	0.960*** (0.082)
June	0.613*** (0.151)	1.01*** (.152)	0.906*** (0.135)	1.54*** (0.132)	0.762*** (0.149)	1.39*** (0.143)	0.751*** (0.122)	0.955*** (0.121)	0.813*** (0.785)	1.27*** (0.081)
July	0.716*** (0.159)	1.17*** (.160)	0.873*** (0.146)	1.56*** (0.143)	0.778*** (0.147)	1.45*** (0.143)	0.701*** (0.122)	0.996*** (0.121)	0.821*** (0.080)	1.35*** (0.083)
August	0.645*** (0.169)	1.04*** (.170)	.886*** (0.166)	1.39*** (0.163)	0.942*** (0.151)	1.48*** (0.145)	0.821*** (.120)	1.06*** (0.119)	0.939*** (0.082)	1.38*** (0.086)
September	0.698*** (0.173)	1.11*** (0.174)	1.01*** (0.180)	1.57*** (0.177)	0.922*** (0.180)	1.51*** (0.174)	0.544*** (.120)	0.803*** (0.119)	0.865*** (0.086)	1.32*** (0.090)
October	0.420** (0.173)	0.574*** (.174)	0.427*** (0.166)	0.778*** (0.163)	0.360** (0.180)	0.781*** (0.174)	0.364*** (0.120)	0.490*** (0.119)	0.535*** (0.086)	0.821*** (0.089)
November	0.257 (0.173)	0.505*** (0.174)	0.372*** (0.139)	0.605*** (0.136)	0.317** (.153)	0.570*** (0.147)	0.219* (0.120)	0.289** (0.119)	0.357*** (0.081)	0.568*** 0.084)
December	−0.291* (0.173)	−0.200 (0.174)	0.159 (0.135)	0.220* (0.132)	.109 (0.147)	0.168 (0.141)	.042 (0.121)	−0.026 (0.120)	0.092 (0.080)	.114 0.083)
Constant	−0.330*** (0.119)	−0.675*** (0.120)	−0.686*** (0.093)	−1.12*** (0.091)	−0.722*** (0.102)	−1.03*** (0.099)	−0.044 (0.092)	−0.110 (0.091)	−0.464*** (0.057)	0.747 (0.059)
N	147	147	192	192	213	213	309	309	861	861
R^2 within									0.319	0.542
R^2 between									0.679	0.621
R^2 overall	0.392	0.588	0.359	0.637	0.331	0.603	0.306	0.459	0.299	0.486
Wald X^2									362.04	802.09

Notes: Standard errors in parentheses. January is the reference month. ***$p < 0.01$, ** $p < 0.05$, * $p < 0.1$. AL = Air Lead, AS = Air Soil.

atmospheric Pb for city j being $\beta_0 + u_j$. The city-specific error component can be thought of as the combined effects of omitted city characteristics or unobserved heterogeneity (Rabe-Hesketh and Skrondal, 2008). The daily station reading residual e_{ij} is the difference between observed atmospheric Pb level on given day i and the average atmospheric Pb of that station's city mean, $e_{ij} = y_{ij} - (\beta_0 + u_j)$ Both residual terms are assumed to obey a Gaussian structure with zero means: $u_j \sim N(0, \sigma_u^2)$ and $u_j \sim N(0, \sigma_u^2)$.

To address the seasonality of atmospheric Pb and soil, and using the same modeling logic above, we regressed both atmospheric Pb and soil on the weather variables of average relative humidity, average sea level pressure, average temperature, average visibility, and average wind speed to derive seasonally-adjusted atmospheric Pb and soil estimates. The weather-adjusted air Pb and soil estimates were then graphed on the daily time interval, fitting the distribution of weather-adjusted daily readings of air Pb and soil with median splines. This analysis was performed for all cities combined, and then for each city independently. The graphical analysis provides a visual check to see if daily variations in weather-adjusted air Pb and soil estimates approximate to several outcomes including: Observed seasonal cycles in the proportion of children screened with blood Pb $> 10\ \mu g\ dL^{-1}$ (Haley and Talbot, 2004); and age-adjusted average blood Pb (USEPA, 1995; Havlena et al., 2009). Finally, weather-adjusted air Pb and soil estimates were regressed on monthly dummy/binary variables to see if coefficients rise noticeably in the months of June, July, August and September, which are known periods when children presented with elevated blood Pb.

To test whether anthropogenic turbulence may drive re-suspension of Pb-contaminated urban soil into the atmosphere we performed independent sample t-tests to compare atmospheric soil and Pb aerosol concentrations on weekdays and weekends/Federal Government holidays. Both industrial activities and vehicle miles traveled decline significantly on weekends and Federal Government holidays mandating closure of businesses including: New Year's Day, Martin Luther King Day, President's Day, Memorial Day, Independence Day, Labor Day, Columbus Day, Veterans Day, Thanksgiving Day, and Christmas Day.

3. Results

3.1. Least squares regression models for each city

Results for ordinary least squares regression models for each city and a random effects generalized least squares regression model combining city observations are suitable for statistical procedures used herein (Table 2). We hypothesized the natural log of air Pb was a function of the natural log of air soil. Marginal effects are reported, indicting the predicted percent change in atmospheric Pb for a percent change in atmospheric soil. Results show that atmospheric Pb and soil are correlated significantly. The expected percent increase in atmospheric Pb for a percent increase of air soil is 0.709 (95% CI, 0.535 to 0.882) in Pittsburgh, 0.848 (95% CI, 0.724 to 0.973) in Detroit, 0.710 (95% CI, 0.573 to 0.847) in Chicago, and 0.922 (95% CI, 0.812 to 1.033) in Birmingham. The soil elasticity or marginal effect of atmospheric Pb is approximately equal across all the cities analyzed, given overlapping intervals of confidence. In the random effects model combining observations from all cities, we find a marginal effect of 0.841 (95% CI, 0.775 to 0.908). Model residuals from the random effects regression have a mean of zero (1.11e−09) and white noise properties of Gaussianity and homoskedasticity. Overall, both within and across the four cities examined, atmospheric Pb and soil co-varied in near proportional terms for the four cities tested.

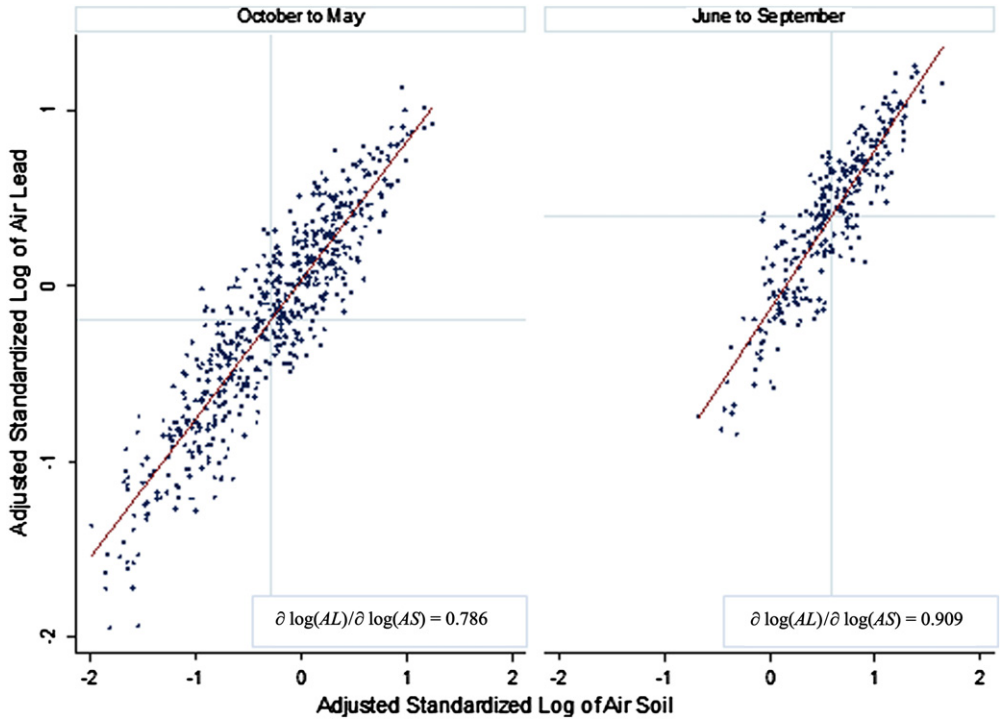

Fig. 4. Weather adjusted air lead versus air soil with linear fit for peak months (June, July, August, and September) and non peak months (October through May) for Pittsburgh, Detroit, Chicago and Birmingham combined.

3.2. Atmospheric soil and Pb aerosols as a function of weather variables

The random effects regression models of atmospheric Pb and soil as a function of weather variables were determined (Table 3). Allowing j to denote city, i to denote the day of the atmospheric Pb or atmospheric soil station reading, and y_{ij} denote the atmospheric Pb of soil level on day i in city j, our regression estimator is modeled as: $y_{ij} = \beta_0 + \beta_1 W_{ij} + u_j + e_{ij}$, where, β_0 is the average air Pb or average air soil across cities, and W_{ij} is a vector of weather variables observed daily and corresponding to a given city. The double component residual of the random effects model obeys the same logic as described above. Both regular (b) and standardized coefficients (beta) are reported. In Model 2, we find that standard deviation increases in humidity (beta = −0.073), visibility (beta = −0.135), and wind speed (beta = −0.393), significantly decrease atmospheric Pb, whereas sea level pressure (beta = .103) and temperature (beta = 0.288) function to increase atmospheric Pb. To be more specific, a standard deviation increase in observed temperature increases the expected level of atmospheric Pb by over 1/4th of a standard deviation. Weather variables behave similarly in direction and magnitude in prediction of atmospheric soil. Adjusting for weather variables, both atmospheric Pb and soil appear sensitive to anthropogenic turbulence. Measured as a binary variable of weekday = 0, and weekend/Federal Government holiday = 1, we find that air Pb (beta = −0.122) and air soil (beta = −0.289) levels are decreased significantly during days of rest and leisure.

Across and within the cities examined, atmospheric soil and Pb appear to have cyclical properties of consistent periodicity, angle function and amplitude, peaking in the summer/autumn months June, July, August and September, and contracting noticeably in the winter months of October through May (Table 3; Figs. 2 and 3). The behavior of both Pb aerosols and air soil splines appear to match closely the cyclical behavior of blood Pb outcomes in children observed across many cities and time periods (Laidlaw et al., 2005).

3.3. Models of weather-adjusted Pb aerosols and air soil values

Regression models of weather-adjusted air Pb and air soil values as a function of monthly dummy variables were also developed (Table 4), revealing that within and across the study cities, air Pb and soil follow a seasonal pattern, which is distinguishable from a chance occurrence. Reported coefficients represent expected increases or decreases in average atmospheric Pb and soil in specified months as compared to our reference months of October through May. Coefficients are expressed in standard deviation terms. For example, in the random effects model, incorporating observations from all cities, we find that in the months of June, July, August, and September, atmospheric soil levels are between 1.27 and 1.38 standard deviations higher than in the months of October through May. Lead aerosol levels are similarly higher in the months June, July, August and September, rising above the October through May levels by 0.813 to 0.939 standard deviations. Again, these summer and autumn months correspond to the same months where the incidence of child Pb poisoning is highest (Laidlaw et al., 2005).

The theoretical expectation is that the relationship between air Pb and air soil ought to strengthen in the peak months of June through September as compared to non-peak months of October through May. Not only do mean values noticeably increase on both dimensions, but the elasticity of the relationship between air Pb and air soil statistically increases ($\partial\log(\mathrm{AL})/\partial\log(\mathrm{AS}) = 0.786$ versus 0.909; Fig. 4). The same pattern is evident in each of the individual cities (Fig. 5). This result of a rising marginal effect in the summer/autumn months of June, July, August, and September is consistent with the soil re-suspension mechanism of atmospheric Pb aerosols.

3.4. Atmospheric soil and Pb aerosol levels on weekdays and weekends/Federal Government holidays

Independent sample t-test results comparing average atmospheric soil and Pb aerosol levels on weekdays and weekends/

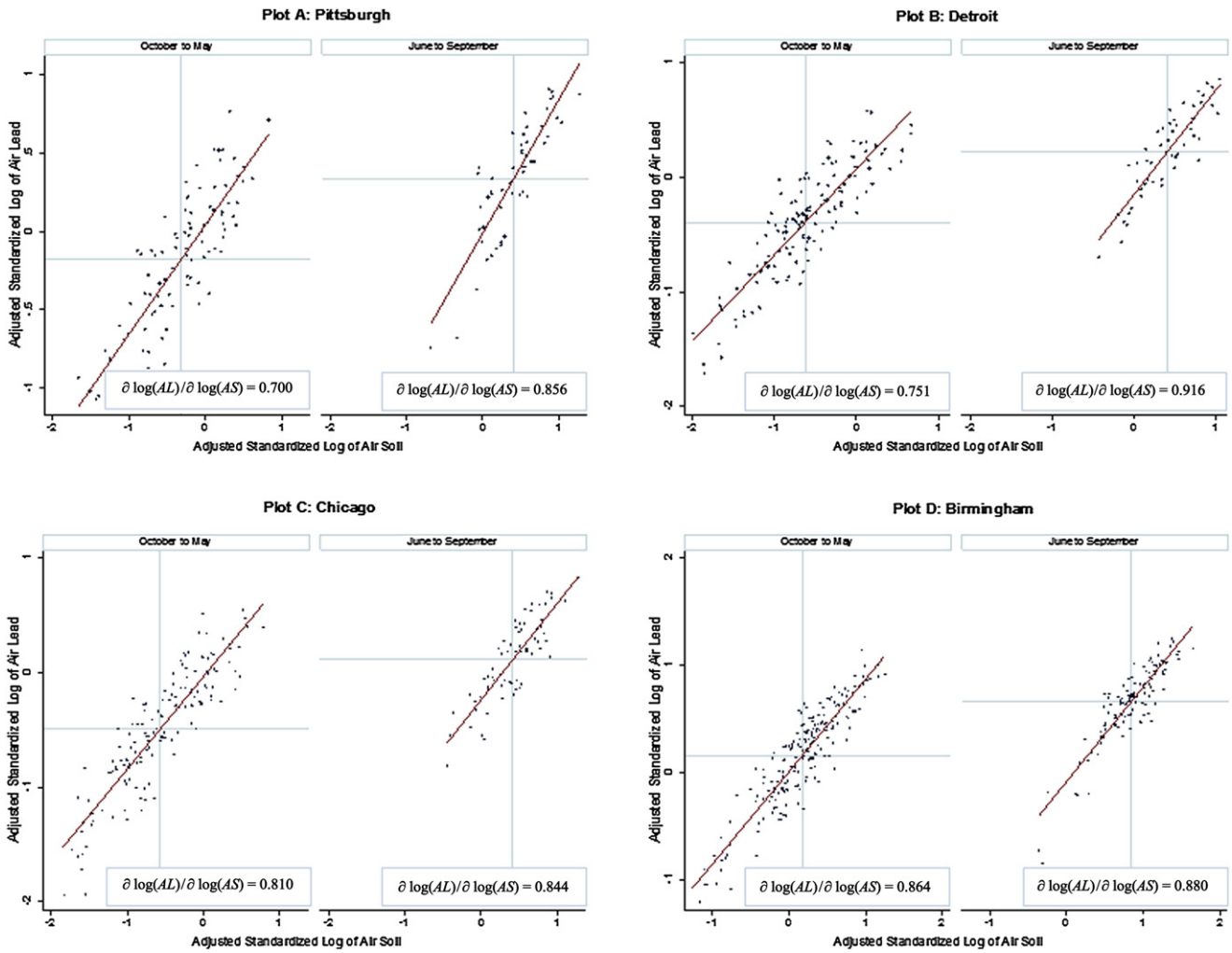

Fig. 5. Weather adjusted air lead versus air soil with linear fit for soil during ail peak months (June, July, August, and September) and non-peak months (October through May) by city, Pittsburgh, Detroit, Chicago and Birmingham.

Federal Government holidays provide an interesting insight into the role of atmospheric turbulence in soil re-suspension (Table 5). The main purpose here is to test whether anthropogenic traffic turbulence influences observed levels of soil and Pb in the atmosphere. By exploiting the weekly and holiday structure of labor and leisure routines in the US, we were able to conduct a natural experiment examining the role of anthropogenic turbulence of soil and atmospheric Pb. Results show that both weather adjusted Pb aerosols ($t = 4.191$, $\Pr(W_d > W_e) = 0.01$) and air soil levels ($t = 6.443$, $\Pr(W_d > W_e) = 0.01$) are significantly lower on weekends/holidays across all cities examined. Moreover, Pb aerosol levels are 3.12 times higher during weekdays than weekends, and air soil levels are 3.15 times higher during weekdays than weekends. These findings are consistent with the concept that anthropogenic turbulence influences both atmospheric soil and Pb aerosol concentrations, which in turn may drive exposure inside residential homes.

4. Discussion

4.1. Study limitations

Atmospheric soil and Pb aerosol concentrations were only observed at one location in each of the cities, and it is not known if these temporal patterns would be confirmed spatially throughout each of the cities. Also, causality between atmospheric soil re-suspension and Pb aerosol concentrations was inferred and could not be proved from this study; although we note that the association is strongly correlated and consistent between the cities examined. The potential health impacts of soil Pb re-suspension as

Table 5
Descriptive statistics and independent samples *t*-test results comparing weekdays and weekends/Federal Government holidays.

	Adjusted standardized log of air lead				Adjusted standardized log of air soil			
	Weekday (W_d) Mean (Std. Error)	Weekend (W_e) Mean (Std. Error)	Difference	*t*-test	Weekday (W_d) Mean (Std. Error)	Weekend (W_e) Mean (Std. Error)	Difference	*t*-test
Overall	0.0569 (0.0240)	−0.1209 (0.0349)	0.1778 (0.0424)	4.191***	0.1037 (0.0288)	−0.2232 (0.0416)	0.3268 (0.0507)	6.443***

Note: ***$\Pr(W_d > W_e) = 0.01$.

indicated by other studies can also only be inferred because childhood blood Pb data was not available for the four cities examined.

4.2. Re-suspension of Pb contaminated soil and vehicle traffic

By definition, atmospheric soil particles have been re-suspended from the ground surface. As observed in multiple locations in the US, atmospheric soil peaks in the summer and autumn during periods of high evapotranspiration potential and low soil moisture (e.g. Laidlaw and Filippelli, 2008; Wells et al., 2007). Given that, as reviewed by Mielke et al. (2011), many urban soils have been contaminated with Pb, it is logical to conclude that when these soils are re-suspended at higher rates during the summertime, atmospheric Pb aerosols will also increase. The contention that Pb contaminated soil is a major source of urban atmospheric Pb, as indicated by the results of this study, is supported by the published literature.

Soil Pb particles are thought to be re-suspended into the atmosphere during the summertime and autumn where they penetrate into the interior of homes and settle on contact surfaces (Layton and Beamer, 2009). Cho et al. (2011) conducted a literature review of the concentrations and size distributions of ambient airborne Pb containing particulate matter, and revealed that during the era of leaded gasoline airborne particle-bound Pb was typically submicron sized whereas after the withdrawal of leaded gasoline, the largest mode of the size distribution of particles shifted to particles > 2.5 μm (typically associated with larger particles such as soil dust). Since the phase-out of leaded gasoline, airborne Pb concentrations have significantly decreased in urban areas. Cho et al. (2011) concluded that "near-roadway dust" or near industrial sources (when applicable) have become the two most important contemporary sources of Pb in the atmosphere. In Baltimore, Maryland, soil re-suspension has also been observed to be higher in the high traffic older urban areas than in the newer low traffic suburban areas (Simons et al., 2007).

International studies also support our findings. In Sydney Australia, Davis and Birch (2011) measured bulk atmospheric Pb deposition near two background sites located at least 500 m from a major roadway and adjacent to three roadways with traffic volumes of 2000, 47,500 and 84,500 vehicles per/day. This study showed that the Pb flux at these sites was 12, 29, 38, 83 and 106 $\mu g\, m^{-2}\, day^{-1}$, respectively, and was also proportional to traffic volume, corroborating Cho et al.'s (2011) review, which also showed that re-suspension of Pb contaminated roadside soils resulted from truck and automobile turbulence on high traffic roadways. In Berlin, Germany, Lenschow et al. (2001) observed that at curb-sides on main streets, the PM10 concentration is up to 40% higher than the urban background; half of the additional pollution is due to motor vehicle exhaust emission and tire abrasion and the other half due to re-suspended soil particles.

Given that aerosols have a high surface area to mass ratio, they are likely to have a relatively high bio-reactivity. Given their small particle size, they also can invade homes and become deposited on horizontal surfaces more easily than coarse particles (Layton and Beamer, 2009). Consequently, seasonal re-suspension of fine particulates from Pb contaminated urban soils may be driving chronically elevated Pb levels in urban children (Harris and Davidson, 2005; Laidlaw and Filippelli, 2008; Laidlaw et al., 2005; Zahran et al., 2011).

Lead contaminated urban soil is implicated as the major source for atmospheric Pb aerosol loadings. A consequence of the re-suspension of Pb contaminated soil is that it has significant repercussions for ongoing adverse Pb exposures in urban dwelling US children. Pingitore et al. (2009) observed that if Pb contaminated urban soil is the principal source for airborne Pb in urban settings, then "*contaminated soil may set a practical lower limit for future decreases in regulation of airborne lead levels*" (Pingitore et al., 2009, p. 5). The Centers for Disease Control and Prevention (CDC) acknowledges that along with Pb-based paint and Pb dust, soil is a major Pb source; the agency does not provide a single recommendation about soil Pb in their guidelines (CDC, 2007).

5. Conclusions

In order to decrease urban atmospheric Pb concentrations, subsequent Pb-rich dust deposition and penetration into homes, and its consequent deleterious effect in childhood Pb levels, it is necessary to remediate and or isolate urban soils contaminated with Pb. While the US Federal Government has enacted legislation covering clean air (USEPA, 2010) and clean water (USEPA Federal Water Pollution Control Amendments of 1972), there is no universal clean soil act, although there are several standards pertaining to acceptable values. These guidelines are inconsistent across the US and in light of the evidence, they need to be harmonized and re-evaluated so as to develop a unified strategy to mitigate an unnecessary and preventable exposure pathway.

Acknowledgements

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Chapter 4 - Paper 2: "*Linking Source and Effect: Re-suspended Soil Pb, Air Pb, and Children's PbB Levels in Detroit, Michigan"*

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Linking Source and Effect: Resuspended Soil Lead, Air Lead, and Children's Blood Lead Levels in Detroit, Michigan

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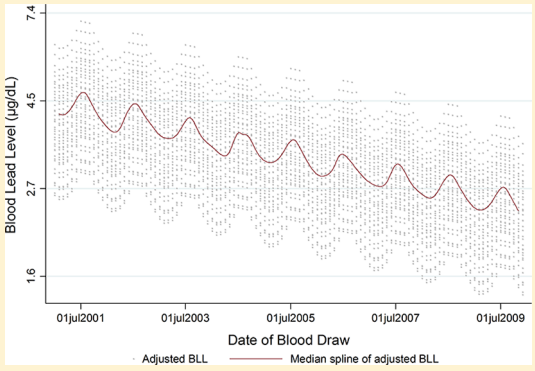
Supporting Information

ABSTRACT: This study evaluates atmospheric concentrations of soil and Pb aerosols, and blood lead levels (BLLs) in 367 839 children (ages 0−10) in Detroit, Michigan from 2001 to 2009 to test a hypothesized soil → air dust → child pathway of contemporary Pb risk. Atmospheric soil and Pb show near-identical seasonal properties that match seasonal variation in children's BLLs. Resuspended soil appears to be a significant underlying source of atmospheric Pb. A 1% increase in the amount of resuspended soil results in a 0.39% increase in the concentration of Pb in the atmosphere (95% CI, 0.28 to 0.50%). In turn, atmospheric Pb significantly explains age-dependent variation in child BLLs. Other things held equal, a change of 0.0069 $\mu g/m^3$ in atmospheric Pb increases BLL of a child 1 year of age by 10%, while approximately 3 times the concentration of Pb in air (0.023 $\mu g/m^3$) is required to induce the same increase in BLL of a child 7 years of age. Similarly, a 0.0069 $\mu g/m^3$ change in air Pb increases the odds of a child <1 year of age having a BLL ≥ 5 $\mu g/dL$ by a multiplicative factor of 1.32 (95% CI, 1.26 to 1.37). Overall, the resuspension of Pb contaminated soil explains observed seasonal variation in child BLLs.



INTRODUCTION

Lead (Pb) remains a serious threat to children's health and development—elevated levels of Pb in the blood are associated with impaired cognitive, motor, behavioral, and physical abilities.[1] Even lead-exposed children with blood lead levels (BLLs) below the World Health Organization (WHO) guideline of 10 $\mu g/dL$ for their entire lifetime experience measurable loss in cognition.[2] In response to health risks associated with BLLs below 10 $\mu g/dL$,[3−5] the U.S. Centers for Disease Control and Prevention (CDC) lowered the blood Pb reference value to 5 $\mu g/dL$ in May 2012.

Average BLLs in the U.S. (and globally) declined following the elimination of Pb from most product streams (e.g., gasoline, paint, water pipes, and solder used to seal canned goods). While airborne Pb used to be extremely high in cities, largely from the direct combustion of leaded gasoline and deposition of Pb oxides, much of the current airborne Pb is from these legacy sources. Contemporary air Pb is in the form of resuspended fine particulates.[6−10]

In this paper, we aim to explain the lingering sources of Pb in Detroit, Michigan by analytically reconciling a compelling empirical fact: *average BLLs for children in the northern hemisphere peak in summer and autumn and retreat during winter and spring periods.*[11] In Detroit, the BLLs of children follow this seasonal phenomenon (see Supporting Information (SI)).

As compared to the reference month of January, child BLLs are found to be between 11% and 14% higher in the months of July, August, and September (described in detail in Figure SI1 and Table SI1 of the SI). The seasonal behavior of child BLLs in Detroit is clear. Explaining this seasonal phenomenon is the aim of our paper, and it is our contention that any theory of contemporary Pb risk must logically account for this striking empirical observation.

Our intuition of what could plausibly account for the seasonality of child BLLs in Detroit is derived from a series of known facts. First, similar to many other postindustrial cities, elevated concentrations of environmental Pb are found

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throughout the Detroit metropolitan area,[12] with especially high concentrations of Pb in soils (400−800 mg/kg) located in the interior of the city[13] that correlate with spatial variation in children's BLLs.[14] Outdoor soil is a reservoir of legacy Pb from multiple anthropogenic sources and may explain why many household intervention efforts are unsuccessful.[15] Second, research shows that contemporary atmospheric concentrations of Pb spike during summer and autumn in many U.S. cities, including Washington, DC,[16,17] Boston,[18] Milwaukee,[19] New York,[20] New Jersey,[21] and Chicago.[22] In fact, seasonal variations in child BLLs and atmospheric Pb are strikingly similar. Third, previous research has demonstrated a remarkable ability to predict child BLLs based on climate variables.[23] Taken together, these facts are suggestive of a soil → air dust → child pathway of contemporary Pb exposure, where Pb-contaminated urban soils are resuspended as dust subject to seasonal precipitation regimes, wind, humidity, and other meteorological factors, with air Pb dust inhaled and ingested by unsuspecting children.

To evaluate this hypothesized pathway, this study uses temporally resolved atmospheric soil and Pb data, and matched BLL data from the Detroit metropolitan area. Statistical and numerical modeling are used to determine correlation strengths across a range of environmental and human variables, and specifically to target the contributions of air Pb to child BLLs likely derived from soil resuspension as opposed to point source air Pb emissions.

MATERIALS AND METHODS

To address the soil → air dust → child pathway for Pb exposure, a number of data sources are examined: blood Pb data for 367 839 children from the Michigan Department of Community Health (MDCH); atmospheric soil and Pb data from the U.S. Environmental Protection Agency's (EPAs) Interagency Monitoring of Protected Visual Environments (IMPROVE) database; [24] local weather data from the National Weather Service, National Climatic Data Center; and point location information on Pb-emitting facilities from the EPA's Toxic Release Inventory.[25]

Blood Pb Data. Children's blood Pb data for the tricounty area encompassing the City of Detroit was obtained from the Michigan Department of Community Health (MDCH). The data set contains blood samples collected from January 2001 through December 2009. Blood Pb measurements are reported as integers in units of micrograms per deciliter of blood (μg/dL). MDCH data also contain information on the census tract residential location of each child, the month and year of sample collection, child age in years (0−10), child sex (male = 1, female = 0), and the blood draw type (1 = capillary, 0 = venous). As with previous research,[26] we analyze child BLLs as a continuous variable (μg/dL) and as dichotomous variable ($\geq$5 μg/dL = 1, < 5 μg/dL = 0).

Atmospheric Pb and Soil Data. Atmospheric soil and Pb aerosol data were obtained from IMPROVE for the period of January 2001 to December 2009 (Station 261630001; additional stations presented in the SI). To derive atmospheric soil estimates, we use a mineral equation based on the elemental composition of soil.[27] Soil composition is derived by the quadratic sum of aluminum (Al), silica (Si), calcium (Ca), iron (Fe), and titanium (Ti) concentrations, assuming independence of measurement uncertainties as described by:

$$[\mathrm{d(soil)}]^2 = [2.20 \times \mathrm{d(Al)}]^2 + [2.49 \times \mathrm{d(Si)}]^2 + [1.63 \times \mathrm{d(Ca)}]^2 + [2.42 \times \mathrm{d(Fe)}]^2 + [1.94 \times \mathrm{d(Ti)}]^2 \quad (1)$$

Both atmospheric soil and Pb aerosol quantities are measured in units of μg/m^3. The quantity of soil derived using this equation is an estimate subject to spatial variability of soil composition and anthropogenic interferences.

Local Weather Data. Given that local weather conditions influence atmospheric concentrations of soil and Pb, we collected data describing the 24 h average relative humidity (%), sea level pressure (mb), temperature (°C), visibility (km), and wind speed (kmph) on the day of atmospheric readings.[6]

Point Source Pb. Under section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA), firms that release, transfer, or dispose of listed toxins are required to submit annual reports to the EPA detailing quantities of toxins emitted. Data are published under the Toxic Release Inventory (TRI) system.[25] Over the period of observation, 2001 to 2009, a total of 22 Pb-emitting facilities operated in Detroit. In analyses that follow, we estimate whether the presence of a point source polluter of Pb in a child's residential zip code predicts BLL outcomes.

Empirical Strategy. First, we analyze the extent to which daily variation in atmospheric Pb is explained by atmospheric soil.[6] The expectation is that atmospheric Pb and soil are statistically correlated, and that weather-adjusted Pb and soil concentrations in the atmosphere have distinct seasonality, rising and falling simultaneously over the calendar year. Insofar as atmospheric concentrations of soil and Pb move together seasonally, the first link in our soil → air dust → child pathway for Pb exposure is statistically corroborated. We use a least-squares regression procedure to examine the association between atmospheric soil and Pb. Formally, letting y_t denote atmospheric Pb in Detroit on day t our regression estimator is modeled as

$$y_t = \beta_0 + \beta_1 S_t + \Gamma_1 W_t + \varepsilon_t \quad (2)$$

where β_0 is the model constant; S_t is the atmospheric soil reading on day t; W_t is a vector of local weather conditions on the day atmospheric Pb and soil are measured, and ε_t is the error term, with $\varepsilon_t \sim IDD\ (0,\sigma_y^2)$. After testing whether atmospheric Pb levels in Detroit are statistically associated with atmospheric soil, we analyze the extent to which variation in child BLLs might be explained by atmospheric Pb concentrations, the second link in our hypothesized pathway.

To determine this relationship, we first analyze child blood Pb as a continuous variable (μg/dL). A census tract fixed effects regression procedure was used to analyze child BLLs. Letting y_{ijt} denote the BLL of child i in census tract j in month t the regression estimator is modeled as

$$y_{ijt} = \alpha_j + \beta_1 L_t + \beta_2 P_j + \Gamma_1 A_i + \Gamma_2 C_i + \Gamma_3 M_i + \Gamma_4 Z_{it} + \varepsilon_{ij} \quad (3)$$

where α_j is the census tract fixed-effect accounting for unobserved heterogeneity at the neighborhood level; L_t is the average monthly weather-adjusted atmospheric Pb level derived from eq 2 (child BLLs are indexed by month, warranting change in the time-step of Pb aerosol data); P_j is a dummy variable equal to 1 if a Pb emitting facility operates in the zip code of a child's residential location; A_i is the age of the child in years; C_i is a dummy variable equal to 1 if the blood draw was

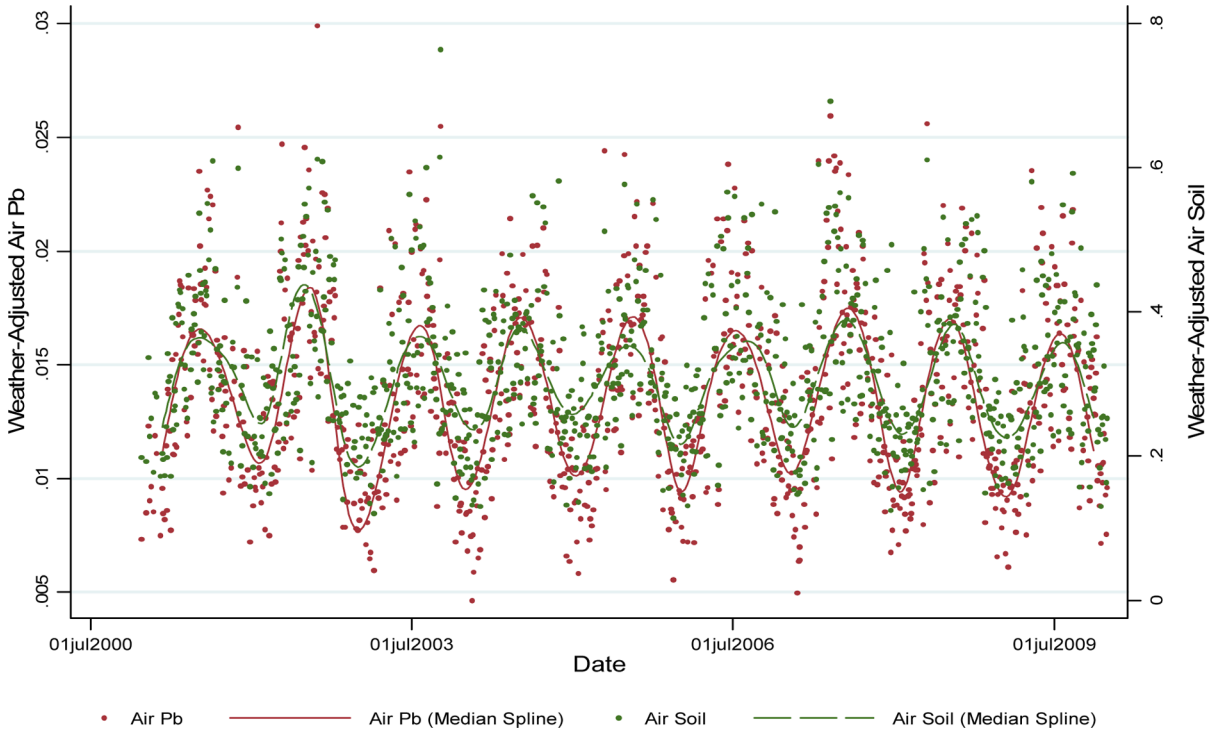

Figure 1. Scatterplot with median splines of weather adjusted air Pb and air soil in time (daily). Weather-adjusted air Pb and soil estimates ($\mu g/m^3$) (Models 3 and 4 described in SI) are graphed on the daily time-step, fitting distributions of air Pb and soil values with median splines. Time is on the x-axis, weather-adjusted atmospheric Pb is on the y_1-axis and atmospheric soil is on the y_2-axis. Air Pb values are in maroon and air soil values are in forest green, with corresponding median splines colored similarly.

capillary; M_i is a dummy variable equal to 1 if the child is male; Z_{it} corresponds to the year the blood draw occurred, and ε_{ij} is the error term, with $\varepsilon_t \sim IDD\ (0,\sigma_y^2)$.

Second, using a conditional fixed effect logistic regression procedure, we analyze whether or not a child's BLL is ≥5 μg/dL (corresponding to the CDCs new reference value) as a function of atmospheric Pb. Letting Y represent the threshold BLL of a child, where Y equals 1 if a child's BLL is ≥5 μg/dL, and Y equals 0 if a child's BLL is <5 μg/dL. We specify the following reduced form logistic equation for the probability of threshold exceedance (Y) for child i in census tract j in month t:

$$\begin{aligned}\text{Prob}(Y_{ijt} = 1|L_t, P_j, A_i, C_i, M_i, Z_{it}) \\ = \Lambda[\alpha_j + \beta_1 L_t + \beta_2 P_j + \Gamma_1 A_i + \Gamma_2 C_i + \Gamma_3 M_i \\ + \Gamma_4 Z_{it}] \end{aligned} \quad (4)$$

where $\Lambda[\cdot]$ is the CDF of the logistic distribution. The definition of other terms carries from eq 3. The theoretical expectation under eqs 3 and 4 is that child BLL outcomes rise with atmospheric Pb.

Statistical models of child BLLs divide children by reported ages in years. Two reasons motivate this decision. First, low gastric exclusion for Pb in children and high dissolution potential of particulates (due to high surface area to mass ratios) are known to elevate BLLs in children in age-dependent ways.[28] Second, based on strong age-related risk factors observed for children,[29] we logically assume that the effects of airborne exposure are best observed in especially young children (ages 0–2) since they are *relatively* immobile and more insulated from other known sources of Pb (e.g., paint chips, direct interaction with Pb contaminated soils). Therefore, inasmuch as the proposed soil → air dust → child pathway for Pb exposure is a plausible description of contemporary Pb risk, coefficients on atmospheric Pb in eqs 3 and 4 ought to be noticeably higher in children less than 2 years of age.

RESULTS AND DISCUSSION

Atmospheric Soil and Pb Relationships. Ordinary least-squares regression models were rendered to predict atmospheric Pb as a function of air soil in Detroit, Michigan from January 2001 to December 2009. Variables are log transformed and elasticities are identified (model details provided in SI). The first model (Model 1) results indicate a 1% increase in atmospheric soil results in a 0.48% (95% CI, 0.38 to 0.58%) increase in atmospheric Pb. The association of air Pb and air soil, adjusting for local weather conditions, is presented as Model 2. Results are similar, with air Pb increasing 0.39% (95% CI, 0.28 to 0.50%) for every 1% increase in atmospheric soil. Standardized betas indicate the atmospheric soil is the strongest predictor of air Pb among variables examined. Note that both atmospheric Pb and soil are similarly sensitive to local weather conditions, rising significantly with average temperature and sea level pressure, and declining significantly with average visibility and wind speed (Models 3 and 4). While traffic-induced resuspension of nonsoil particles (derived from wheel weights, brake pads, etc.) is known to be a significant source of atmospheric particulate matter and metals,[30] including a proxy for this variable did not alter the effect observed from atmospheric soil (detailed discussion provided in SI Table SI2).

As shown in Figure 1, weather-adjusted air Pb and soil estimates (derived from Models 3 and 4) have remarkably similar cyclical properties of periodicity, angle function, and amplitude, peaking in the summer/autumn months of June, July, August, and September, and contracting noticeably in the

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Table 1. Conditional Fixed Effect Logistic Regression Odds Ratios Predicting Blood Pb ($\geq$ 5 μg/dL) in Detroit Children, 2001−2009[a]

	age 0 odds ratio	age 1 odds ratio	age 2 odds ratio	age 3 odds ratio	age 4 odds ratio	age 5 odds ratio	age 6 odds ratio	age 7 odds ratio	age 8−10 odds ratio
air Pb	1.316***	1.251***	1.177***	1.116***	1.093***	1.128***	1.111***	1.076***	1.089***
	(0.027)	(0.011)	(0.011)	(0.0098)	(0.010)	(0.014)	(0.020)	(0.030)	(0.026)
Pb facility	1.052	1.096**	1.123***	1.141***	1.076	0.893	0.911	0.804	1.057
	(0.091)	(0.043)	(0.050)	(0.048)	(0.046)	(0.054)	(0.077)	(0.100)	(0.109)
capillary draw	1.969***	1.590***	1.678***	1.909***	2.011***	1.823***	1.895***	1.394***	2.415***
	(0.083)	(0.029)	(0.037)	(0.041)	(0.045)	(0.056)	(0.084)	(0.136)	(0.257)
male	1.066	1.052***	1.197***	1.169***	1.225***	1.262***	1.304***	1.427***	1.311***
	(0.043)	(0.017)	(0.022)	(0.021)	(0.022)	(0.033)	(0.047)	(0.082)	(0.064)
year	0.877***	0.860***	0.833***	0.833***	0.827***	0.810***	0.813***	0.800***	0.798***
	(0.007)	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.006)	(0.010)	(0.008)
N	19,046	75,852	58,322	66,288	66,862	33,878	18,571	8,280	13,122
log likelihood	−7328.2	−42312.1	−33543.5	−37095.8	−35683.7	−17420.0	−8752.6	−3455.4	−5050.8
χ^2	820.8	3684.2	3689.1	4076.6	4132.8	2297.2	1070.8	442.3	658.0
$N_{\text{census tracts}}$	316	370	354	351	350	332	308	287	285

[a]Standard errors in parentheses; ***p < 0.01, **p <0.05.

winter months of December and January. The behavior of both Pb and soil aerosol splines parallel the known cyclical behavior of blood Pb outcomes in children observed across many cities and time periods (details of cyclical behavior presented in SI Table SI1 and Figure SI1).[23]

Next, weather-adjusted air Pb and soil estimates are regressed on monthly dummy variables to observe more precisely how much coefficients rise in the months of July, August, and September. Consistent with findings of Laidlaw et al.,[6] compared to the reference of January, atmospheric Pb levels are 35.7% (95% CI, 28.8 to 42.6%) higher in July; 44.8% (95% CI, 38.0 to 51.7%) higher in August; and 40.0% (95% CI, 33.3 to 47.4%) higher in September. Air soil levels also significantly rise in the months of July (57.8%), August (62.2%), and September (54.0%) as compared to the winter month of January (Model 3, SI Table SI4). Overall, atmospheric Pb and soil have remarkably similar seasonal structure, corroborating the claim that a major source of atmospheric Pb is dust that is resuspended from Pb contaminated urban soils.

Atmospheric Pb and Blood Pb Relationships. Next, we turn attention to the second link in our hypothesized soil → air dust → child pathway. The natural log of child blood Pb is regressed on air Pb, controlling for child sex, blood draw type, and year of observation (statistical details provided in SI Table SI5). Neighborhood (census tract) fixed effects are incorporated into regression results. Recall that these results are grouped by child age. Log transformation of child blood Pb was necessary given high positive skew (5.87) and kurtosis (164.35), effectively eliminating the skew (0.32) and minimizing kurtosis (2.92). An increase of one standard deviation in weather-adjusted air Pb, ~0.0006 μg/m^3, induces an 8.04% (95% CI, 7.1 to 9.0%) increase in blood Pb for children less than 1 year of age. Air Pb significantly increases blood Pb in all children regardless of age, but the increase in child blood Pb for an equivalent unit of air Pb declines noticeably in age. For instance, while it takes approximately 0.0069 μg/m^3 of atmospheric Pb to increase child blood Pb by 10% in children 1 year of age, it requires about 3 times the amount (0.023 μg/m^3) of air Pb to induce a 10% increase in child blood for children 7 years of age.

Interestingly, we also observe an age-specific pattern on differences between males and females in average blood Pb levels (SI Table SI5). No statistically significant difference exists between males and females for children less than 1 year of age. At 1 year of age, a statistically significant difference between boys and girls is apparent and this difference rises incrementally with age. For instance, other things held equal, BLLs in males are only 1.5% higher than females at age 1, but 11.2% higher at age 7. Also worth noting, the effect of a Pb emitting facility in a child's zip code of residence behaves inconsistently by age grouping. For children ages 1−4, we find that residential proximity to a Pb emitting facility increases a child BLL by 2.5−5.5%, depending on the age of the child. For all other ages, proximity to a Pb facility has an effect indistinguishable from chance.

Next, conditional fixed effects odds ratios predicting the expected change in the likelihood of a child's BLL exceeding 5 μg/dL are reported in Table 1. A one standard deviation change in air Pb increases the odds of children less than 1 year of age recording a BLL $\geq$ 5 μg/dL by a multiplicative factor of 1.32 (95% CI, 1.26 to 1.37). Again, the deleterious effect of air Pb declines with age. By comparison, for 7 year olds, the probability of having a BLL $\geq$ 5 μg/dL increases 7.8% (95% CI, 2.0 to 13.8%) for an analogous 0.0006 μg/m^3 increase in air Pb. As with our linear model, residential proximity to a point source polluter of Pb is a significant predictor of whether a child's BLL is $\geq$5 μg/dL for children ages 1−3 only.

Figure 2 graphically illustrates the age-dependent association between blood Pb outcomes and air Pb. Overall, results corroborate our intuition that the effects of airborne exposure are most pronounced in younger children (ages 0−2) that are relatively insulated from other sources of Pb (e.g., paint chips, soil Pb).

The age-dependent association between child BLL and air Pb is further visualized by examining the seasonal behavior of age-stratified blood Pb levels and air Pb in time (Figure 3). Three items are notable. First, regardless of age group, average monthly child BLLs in Detroit are definitively seasonal, rising during the summer period and contracting in the winter period. Second, and again regardless of age group, the rise and fall of child BLLs correspond statistically to the behavior of atmospheric Pb. Third, the blood Pb responsiveness to air Pb behaves noticeably differently by age. For children less than 2 years of age, the rise and fall in blood Pb is more congruent

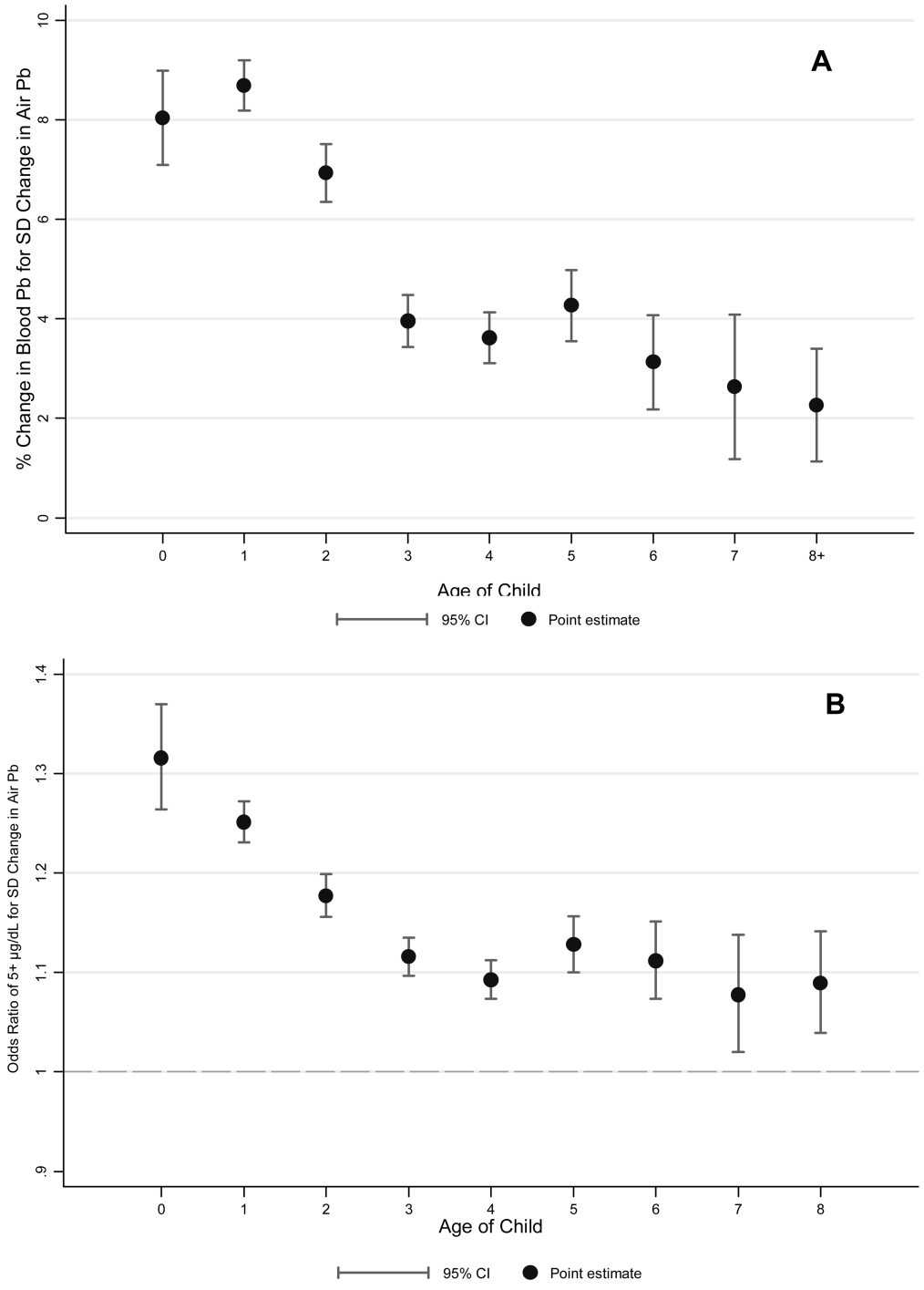

Figure 2. Change in blood Pb outcomes (A) and odds ratio of a 5 μg/dL increase in BLL (B) for standard deviation change in air Pb by age (error bars are 95% confidence intervals).

with the seasonal behavior of atmospheric Pb. To be more precise, the model fit of average monthly blood Pb as a function of air Pb is substantially higher for children age 0−2 as compared to children 6 years of age and older (R^2 = 0.706 vs 0.642). Similarly, a standard deviation rise in atmospheric Pb induces a 0.232 μg/dL (95% CI, 0.203 to 0.260 μg/dL) increase in the monthly average blood Pb of children age 0−2 as compared to a 0.152 μg/dL (95% CI, 0.130 to 0.173 μg/dL) increase in children ≥6 years of age.

Taken together, regression results on child BLL outcomes as a function of atmospheric Pb described in Table 1 and graphically summarized in Figure 3 corroborate the last link in our hypothesized soil → air dust → child pathway for contemporary Pb risk.

As a logical check on this hypothesized pathway, we end by considering a statistical counterfactual. We use the term counterfactual conventionally, meaning to imagine an outcome with modification of an antecedent. The antecedent that we logically modify is the soil source of air Pb. By regressing child BLL on the average monthly residual of eq 2, we have a test of the effect of air Pb on child BLLs absent soil resuspension. That is, the residual in eq 2 constitutes atmospheric Pb with

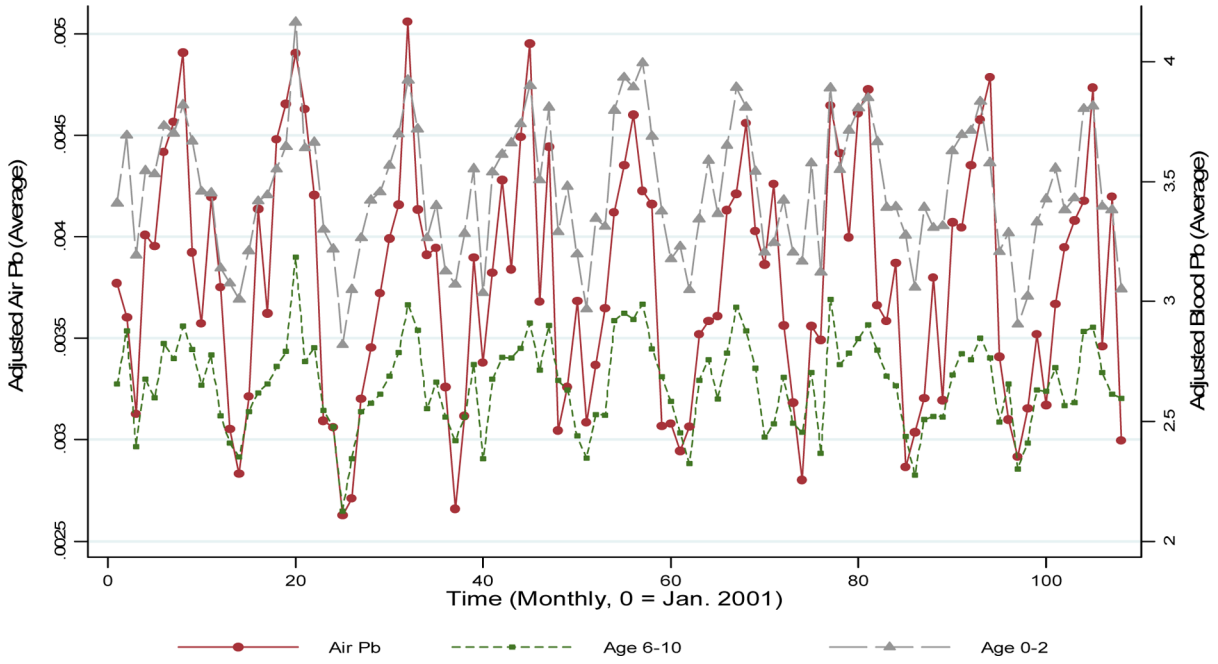

Figure 3. Weather-adjusted air Pb ($\mu g/m^3$) and blood Pb ($\mu g/dL$) by age group. Average monthly child blood Pb levels adjusted by local weather conditions, child gender, method of blood draw, and census tract fixed effects; air Pb estimates are from Model 2 (SI Table SI3).

underlying variation attributable to resuspended soil statistically removed. While the specific content of the residual is unknown, it reflects other unmeasured sources of air Pb; for example, Pb in paint generated by activities such as sanding of home exteriors. Such a test, in effect, interrupts the first link in our causal sequence. Substituting then L_t in eq 4 for ε_t in eq 2, creates then L_t in eq 4 for ε_t in eq 2, creates this counterfactual exercise (results presented in SI Table SI6). With the exception of children age 3, absent soil resuspension, we find that air Pb has an effect on child BLLs indistinguishable from zero. With the soil source of air Pb removed, air Pb has no observable effect on child BLLs—our casual sequence logically disappears.

Overall, empirical analyses corroborate the hypothesized soil → air dust → child pathway for Pb exposure in Detroit children. The data from this study show that daily variation in atmospheric Pb is associated statistically with daily variation in atmospheric soil, with both air Pb and soil showing remarkably similar seasonal properties that match known/observed seasonal variation in child BLLs. In addition, the data demonstrate that air Pb is a significant correlate of child BLLs regardless of age. As expected, and consistent with prior research, the association between child BLLs and air Pb is especially pronounced for children less than 2 years of age.[31] The main exposure mechanism for these young children is likely inadvertent ingestion of fine particulates through hand-to-mouth behavior, exacerbated by poor gastric exclusion for Pb and behavioral patterns that increase surface contact by hand (e.g., crawling). Direct pulmonary uptake remains an untested alternative for some portion of the BLL response. Results from our statistical counterfactual exercise show that absent soil resuspension, the effect of atmospheric Pb on child BLLs is indistinguishable from chance.

The air → dust → child exposure pathway described here may be observed in other urban areas where the legacy Pb deposition in soils remains a critical environmental burden to human health. Our findings suggest that the federal government's continued emphasis on Pb-based paint may be out-of-step (logically) with the evidence presented and an improvement in child health is likely achievable by focusing on the resuspension of soil Pb as a source of exposure. Given that current education has been found to be ineffective in reducing children's exposure to Pb,[15] we recommend that attention be focused on primary prevention of Pb contaminated soils.

■ ASSOCIATED CONTENT

Supporting Information

Sample statistics including details of regression model coefficients and a discussion of the appropriateness of spatial and temporal resolution. This material is available free of charge via the Internet at http://pubs.acs.org.

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The manuscript was written through equal contributions of all authors. All authors have given approval to the final version of the manuscript.

Notes

The authors declare no competing financial interest.

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Chapter 4 - Paper 3: "*Advancing upstream prevention in New Orleans, Louisiana, USA. Determining the relative importance of soil sample locations to predict risk of child Pb exposure."*

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Determining the relative importance of soil sample locations to predict risk of child lead exposure

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ABSTRACT

Soil lead in urban neighborhoods is a known predictor of child blood lead levels. In this paper, we address the question where one ought to concentrate soil sample collection efforts to efficiently predict children at-risk for soil Pb exposure. Two extensive data sets are combined, including 5467 surface soil samples collected from 286 census tracts, and geo-referenced blood Pb data for 55,551 children in metropolitan New Orleans, USA. Random intercept least squares, random intercept logistic, and quantile regression results indicate that soils collected within 1 m adjacent to residential streets most reliably predict child blood Pb outcomes in child blood Pb levels. Regression decomposition results show that residential street soils account for 39.7% of between-neighborhood explained variation, followed by busy street soils (21.97%), open space soils (20.25%), and home foundation soils (18.71%). Just as the age of housing stock is used as a statistical shortcut for child risk of exposure to lead-based paint, our results indicate that one can shortcut the characterization of child risk of exposure to neighborhood soil Pb by concentrating sampling efforts within 1 m and adjacent to residential and busy streets, while significantly reducing the total costs of collection and analysis. This efficiency gain can help advance proactive *upstream, preventive methods* of environmental Pb discovery.

1. Introduction

During the 20th century, lead (Pb) was widely used as a constituent in commercial products, including canned goods, plumbing, folk remedies, lead-based paints and gasoline. With respect to U.S. paint and gasoline, an estimated twelve million metric tons of Pb was used (Clark et al., 1991; Mielke and Reagan, 1998). The legacy of Pb used is reflected in the accumulation of Pb in urban soils. Soil integrates all dust sources of Pb including lead-based paint (either deteriorated, haphazardly removed by power sanding, sand blasted, scraped without capture, or released by building demolition), lead additives in vehicle fuel emissions, and incinerator or industrial Pb emissions (Farfel et al., 2005; Mielke, 1999; Mielke and Reagan, 1998; Mielke et al., 2011a; Rabito et al., 2007). While prevention is the key for protecting children from environmental toxins (Lanphear et al., 2005), concern has been raised about the effectiveness of traditional intervention methods which focus on household environments for reducing children's blood Pb (Yeoh et al., 2012). A major purpose of this study is to evaluate a process for economically discovering community Pb contamination in a manner that supports proactive intervention and prevents childhood Pb exposure.

Soil Pb at or near the surface is an exposure risk to humans through direct contact or re-suspension of Pb in contaminated soils during summer periods (Filippelli et al., 2005; Laidlaw et al., 2005, 2012; Reagan and Silbergeld, 1990; Zahran et al., 2013). Soil lead as a cause for community health concern has been documented by many empirical studies showing strong associations between neighborhood soil Pb, children's blood Pb, and learning or behavioral outcomes (Johnson and Bretsch, 2002; Mielke et al., 1997, 2007; Zahran et al., 2011).

Given that soil Pb is recognized as an important source and predictor of child blood Pb, and assuming that environmental scientists interested in the question of soil Pb risk have fixed budgets, an important soil sampling question arises: *Given scarce resources, where should scientists concentrate soil sample collection efforts to efficiently predict children at-risk for Pb exposure?* To pursue this question of efficient sampling of the soil

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environment, two metropolitan New Orleans datasets were analyzed; one with over 5000 surface soil samples measured for Pb content and stratified by census tracts (Mielke et al., 2005), and the other with geo-referenced blood Pb data for over 55,000 children also stratified by census tracts.

The conventional approach for detecting Pb in the lived environment of at-risk children is by application of a *medical model,* involving routine measurement of children's blood for Pb content. This approach can be characterized as a *downstream methodology* of environmental Pb discovery. If a child records elevated Pb in their bloodstream, then parents, guardians, and authorities follow-up with a search of the lived environment of the child for the source of exposure. By investigating the question of how to efficiently sample neighborhood soils to characterize Pb risk, our study helps advance *upstream or preventive methods* of environmental Pb discovery. In May 2012, the U.S. Centers for Disease Control and Prevention restated a conclusion reached in 1991 that *there is no known safe level of lead exposure* (U.S. CDC Advisory Committee, 2012; U.S. CDC Response to Advisory Committee, 2012; U.S. DHHS, 2012). With no safe level of lead exposure, our investigation can help economize urban soil sampling and mapping efforts to anticipate interventions that minimize the health and developmental costs of elevated blood Pb in children.

2. Materials and methods

2.1. Soil lead (Pb) data

The soil Pb dataset was assembled from samples collected from the upper 2.5 cm of the soil surface within residential metropolitan New Orleans (Mielke et al., 2005). The soil samples were stratified by the census tracts of metropolitan New Orleans (n = 286) (U.S. Census Tracts and Block Number Areas, 1993). Because of relative uniformity in population size and demographic composition, census tracts (also known as enumeration districts) are a sensible geo-statistical unit for describing neighborhood conditions.

Critical to this study is that within each census tract 19 soil samples were systematically collected from *four location types*: within 1 m of home foundations, within 1 m of busy streets, within 1 m of residential streets, and open spaces (i.e., away from roadways and buildings such as parks or large residential lots). Home foundation samples reasonably approximate lead-based paint risk, particularly exterior paint, *as well as* aerosolized Pb deposited in soil next to home foundations. Pb contamination at other soil sample *locations*: busy streets, residential side streets, and open spaces are more likely sourced by leaded vehicle fuel, but also integrate Pb from other media, including lead-based paint. Exposure to lead-paint within homes, in the form of large chips or house dust, as well as outside of homes during demolitions are also well documented sources of exposure (Jacobs and Nevin, 2006; Levin et al., 2008; Rabito et al., 2007).

Extraction procedures for soil sample analysis involved room temperature leachate methods using 1 M nitric acid (HNO_3), a method that correlates well with total methods (Elias et al., 1996; Mielke et al., 1983). This method is safer, faster and lower cost per sample compared with those methods using boiling, concentrated HNO_3. The extraction protocol requires mixing 0.4 g of dried and sieved (#10 USGS <2 mm) soil with 20 ml of 1 M HNO_3 followed by slow agitation on an Eberbach shaker for 2 h at room temperature (~22 °C). The extract is then centrifuged (10 min at 1600 ×g) and filtered using Fisherbrand® P4 paper. The extract is stored in 20 ml polypropylene vials until analyzed. A Spectro Analytical Instruments CIROS® CCD Inductively Coupled Plasma Atomic Emission Spectrometer (ICP-AES) is used to analyze the Pb in each sample. Quality assurance and quality control (QA/QC) is accomplished by calibrating the ICP-AES with certified U.S. National Institute of Standards and Technology (NIST) traceable standards. For each run verification includes the following QA/QC actions at the rate of 1 per 20 samples: The NIST standard (0 and 5 μg/mL) at the beginning and then every 20 samples, calibration verification standards (1 and 10 μg/mL NIST traceable standards), an in-house reference inserted for analysis, a duplicate sample, and a sample blank were also included. The in-house reference (Pb content ~170 mg/kg) is from New Orleans City Park. If verification results differ by >10% the sample run is repeated. The final soil Pb database is the result of the analytical results minus the sample blanks for each soil sample collected in each census tract of metropolitan New Orleans (Mielke et al., 2005).

Overall, the soil survey resulted in 5467 surface samples collected at the rate of ~19 samples from each of the 286 census tracts (Mielke et al., 2005). The neighborhood soil Pb data are summarized as the median value by sample location from home foundations, busy streets, residential streets and open spaces per census tract expressed in mg/kg units. In addition to taking the median of soil Pb samples within each census tract, we operationalized the risk of soil Pb exposure by: (1) taking the mean of soil samples within census tracts, (2) calculating a distance weighted mean soil Pb risk for each child, leveraging the residential location of each child and soil sample location, and (3) interpolating the average soil Pb content within a census tract by ordinary Kriging. All three methods generated highly correlated results (r = 0.89 to 0.96) with the median of soil Pb samples within the census tract performing best in children's blood Pb prediction models.

In the analyses outlined below we aim to discover which neighborhood *soil sample location* best predicts variation in child blood Pb. Descriptive statistics on Pb by soil sample location type are summarized in Table 1.

2.2. Blood lead (Pb) age, and sex data

Blood Pb data for New Orleans were collected and organized by the Louisiana Healthy Homes and Lead Poisoning Prevention Program, 2011 (LAHHLPPP). Details of the childhood blood Pb surveillance system are provided in the LAHHLPP report that documents the details of the combined datasets of both public health and private lab data used to monitor exposures. Medical personal are strongly encouraged to report all children's blood Pb samples to the LAHHLPPP. The datasets were obtained through a formal application by the authors to the LAHHLPPP and individual children are not identifiable in the data set.

Each blood Pb sample was geocoded and matched to the boundaries of the corresponding 1990 census tract. Blood Pb values are expressed in μg/dL units. Blood Pb for 55,551 children from the years 2000-late August, 2005 were included in this analysis. In addition to blood Pb results, the LAHHLPPP data contain information on the age and the sex of the child, as well as the year the blood sample was taken. These variables are included as control variables in statistical models. Residential tenure data from the 2000 Population and Housing Census (item PCT49) show that 85.42% of the population in Orleans Parish had the same address or resided in the same parish from 1995 to 2000, so that age of the child may be conceived as an adequate (though imperfect) surrogate for length of exposure (Zahran et al., 2011).

2.3. Statistical procedures

To analyze blood Pb in children (μg/dL) as a function of location types of soil lead exposure, we used a *generalized least squares random effects regression* where *j* denotes a census tract neighborhood, *i* denotes

Table 1
Descriptive statistics on Pb levels in soil (mg/kg) by location type.

Variable	$P_{.01}$	$P_{.05}$	$P_{.10}$	$P_{.25}$	$P_{.50}$	$P_{.75}$	$P_{.90}$	$P_{.95}$
Busy Street	5.1	11.0	20.8	57.7	156.1	413.9	765.0	1156.1
Foundation	3.2	6.2	8.9	28.5	136.6	1289.0	4637.7	9236.0
Open Space	4.0	6.9	9.6	23.4	71.1	300.0	870.2	1490.0
Residential Street	5.0	9.6	14.9	38.2	104.7	325.4	804.0	1365.0

a sampled child, and y_{ij} denotes the blood Pb level of child i in census tract j. Our regression model is:

$$y_{ij} = \beta_0 + \beta_1 B_j + \beta_2 F_j + \beta_3 O_j + \beta_4 S_j + \Gamma_1 D_i + \Gamma_2 T_i + u_j + e_{ij} \quad (1)$$

where, β_0 is the average blood Pb of children across neighborhoods, and B_j is the busy street soil Pb level in a neighborhood, F_j is the home foundation soil Pb level in a neighborhood, O_j is the open space soil Pb content in a neighborhood, S_j is the residential street soil Pb quantity in a neighborhood, D_i is vector of child demographic characteristics, and T_i is the year the blood sample was taken from child i. The random effects model divides the residual term in two parts: (i) a census tract-specific error component, given by u_j; and (ii) a child-specific error component, which varies between children and census tract, given by e_{ij}. The neighborhood level residual u_j is the difference between census tract j's child blood Pb mean and the overall mean, with the mean child blood Pb for census tract j being $\beta_0 + u_j$. The census tract-specific error component is meant to capture the combined effects of omitted census tract characteristics or unobserved heterogeneity like age of housing stock or socioeconomic conditions (Rabe-Hesketh and Skrondal, 2008). The child-specific residual e_{ij} is the difference between observed blood Pb level of child i and the average blood Pb of children sharing census tract j, where $e_{ij} = y_{ij} - (\beta_0 + u_j)$ Both residual terms are assumed to be Gaussian with zero means: $u_j \sim N(0,\sigma_u^2)$ and $e_j \sim N(0,\sigma_u^2)$.

To estimate the likelihood of a child's blood Pb exceeding 5, 10, 15, or 20 μg/dL as a function of soil Pb location types, we use a *random intercept logistic regression*. The random intercept model relaxes the assumption of conditional independence in the standard logistic regression by adding a census tract-specific random intercept (ζ_j) to the binary prediction equation. Unobserved characteristics at the neighborhood level are integrated in ζ_j, provided $\zeta_j \mid x_{ij} \sim N(0,\psi)$ and i are independent across tracts j, and assuming that, given $\pi_{ij} = \Pr(y_{ij} \mid x_{ij,j})$, y_{ij} are independently distributed as $y_{ij} \mid \pi_{ij} \sim$ *binomial* $(1,\pi_{ij})$. The model is defined as:

$$\text{logit}\left\{\Pr\left(y_{ij}=1 \mid x_{ij},\zeta_j\right)\right\} = \beta_0 + \beta_1 B_j + \beta_2 F_j + \beta_3 O_j + \beta_4 S_j + \Gamma_1 D_i + \Gamma_2 T_i + \zeta_j \quad (2)$$

with, all other terms defined as in Eq. (1).

To statistically assess the relative importance of each soil Pb location type in predicting variation in child blood Pb, we use a variance decomposition analysis. The relative importance soil Pb location types is determined by averaging semi-partials obtained for each predictor across all $p!$ orderings (Zahran et al., 2011). This procedure is specifically designed for statistical problems with co-linear predictors. We detail the logic of our variance decomposition procedure. For the squared multiple correlation coefficient for p predictors, $R^2_{0.123\ldots p}$, the pth squared semi-partial correlation coefficient is given by

$$r^2_{p.123\ldots p-1} = R^2_{0.123\ldots p} - R^2_{0.123\ldots p-1}. \quad (3)$$

Additionally, $R^2_{0.123\ldots p}$ can be defined in terms of squared semi-partial correlation coefficients as

$$R^2_{0.123\ldots p} = \frac{1}{p}\sum_{i=1}^{p}\sum_{j=1}^{p} V_{ij} \quad (4)$$

where

$$V_{ij} = \frac{1}{\binom{p-1}{j-1}} \sum_{\mathcal{A}\setminus\{i\}} r^2_{0(i.\mathcal{A})} \quad (5)$$

and $\mathcal{A}$ $\{1,\ldots,p\}$ is a subset of 1, ..., p containing a combination of $j-1$ predictor indices. Thus, $R^2_{0.123\ldots p}$ is decomposable into $p(2^{p-1})$ squared semi-partial correlation coefficients. The relative importance or contribution to explained variance of soil Pb location predictor i, $i = 1, \ldots p$, is given by

$$C_i = \frac{1}{p}\sum_{j=1}^{p} V_{ij} \times \quad (6)$$

And, since

$$R^2_{0.123\ldots p} = \sum_{i=1}^{p} C_i, \quad (7)$$

the C_i values constitute exhaustive partitions of $R^2_{0.123\ldots p}$. The derived C_i values corresponding to each soil Pb location type constitute the average of semi-partials obtained for each soil Pb location type across all $p!$ orderings of predictors. By dividing individual C_i values by the sum of C_i's, one can determine the proportion of explained variance in a fully specified model attributable to each soil Pb location type (busy streets, home foundations, open spaces, and residential streets), allowing one to order soil Pb location types by their predictive utility.

3. Results

Table 2 shows the descriptive statistics on the average blood Pb of children residing in neighborhoods of either high ($>P_{0.50}$) or low ($<P_{0.50}$) measured soil Pb in busy street, home foundation, open space, and residential street soil sample location types, as well as different combinations of all types. Moving down the principal diagonal, we show the unconditioned average blood Pb levels of children residing in high traffic busy street soil Pb quantity ($\mu = 7.175$ μg/dL, $\sigma = 6.153$ μg/dL) to low traffic residential street Pb quantity ($\mu = 3.995$ μg/dL, $\sigma = 3.038$ μg/dL). The importance of soil location Pb risk, a more telling feature of Table 2, involves joint conditions where one type of neighborhood soil Pb is high and another is low (see also Mielke et al., 2013).

For instance, among the 27,758 children residing in census tracts with high residential foundation soil Pb, reflecting the high risk of exterior paint Pb exposure, we observed an average child blood Pb level of 7.172 μg/dL. By contrast, for the 27,787 children living in neighborhoods with low home foundation soil Pb risk, average blood Pb was 41.7% lower at 4.18 μg/dL. By combining the neighborhood condition of high foundation Pb with low residential street Pb risk, we observe an average blood Pb of 4.671 μg/dL in 3624 children, inducing a near 35% decline over the unconditioned high soil foundation risk scenario. Similarly, by combining low foundation soil risk with high residential street Pb risk, we find that average blood Pb in 3583 children living in such neighborhoods is 6.109 μg/dL, constituting a 46.2% increase over the unconditioned low foundation risk scenario. Of the four soil location types, residential street Pb seems to exercise the greatest influence over average children's blood Pb.

To extend the suggestive results in Table 2, we have reported findings from our random effects regression analyses. In Table 3, we report a series of models. To account for skew in soil Pb predictors, and to approximate the known diminishing return dose–response curve reported elsewhere (Mielke et al., 2007; Zahran et al., 2011), we regress child blood Pb on the square root of Pb in each soil location type. For ease of interpretation, coefficients are expressed in standard deviation terms. In Model 1, child blood Pb is regressed on the time trend variable, showing that child blood Pb in New Orleans has declined over time ($b = -0.094$, $p < 0.001$). In Model 2, the demographic variables of sex and age were added. As observed in prior studies (Zahran et al., 2011), male children have higher blood Pb than females ($b = 0.285$, $p < 0.001$), and blood Pb rises with age ($b = 0.624$, $p < 0.001$). In Model 3, child blood Pb is regressed on soil Pb location types. Soil Pb location type coefficients express the expected change in children's

Table 2
Average child blood Pb (***μg/dL***) by levels soil Pb (High vs. Low) and by soil location type.

	High busy street	Low busy street	High foundation	Low foundation	High open space	Low open space	High residential street	Low residential street
High busy street	7.175		7.558	5.178	7.509	5.529	7.568	4.483
	(6.153)		(6.367)	(4.375)	(6.290)	(5.120)	(6.339)	(3.692)
	26,914		22,580	4334	22,368	4546	23,485	3429
Low busy street		4.169	5.232	3.997	5.212	3.983	6.210	3.906
		(3.294)	(4.288)	(3.069)	(4.205)	(3.066)	(5.046)	(2.891)
		27,144	3779	23,359	4110	23,028	3101	24,037
High foundation			7.172		7.449	5.513	7.548	4.671
			(6.132)		(6.253)	(5.041)	(6.353)	(3.473)
			27,758		23,789	3969	24,134	3624
Low foundation				4.180	5.039	4.043	6.109	3.894
				(3.335)	(4.009)	(3.194)	(4.801)	(2.954)
				27,787	3820	23,967	3583	24,204
High open space					7.116		7.481	4.563
					(6.050)		(6.263)	(3.301)
					27,609		24,154	3455
Low open space						4.252	6.557	3.915
						(3.553)	(5.635)	(2.990)
						27,936	3563	24,373
High residential							7.362	
							(6.193)	
							27,717	
Low residential								3.995
								(3.038)
								27,828

Note: Statistics reported include mean blood Pb level, then standard deviation in parentheses, and then count of children observed.

blood Pb by a standard deviation increase in the square root of soil Pb (mg/kg). As suggested in Table 2, results from Model 3 indicate that residential street soils more strongly predict child blood Pb than foundation soils, busy street soils, and open space soils. Model 4 is our fully saturated model of soil Pb location sources and control variables. Other things held equal, we find that a standard deviation increase in busy street soil Pb, increases the expected child blood Pb by 0.291 μg/dL By contrast, a standard deviation increase in residential street soil Pb increases the expected level of child blood Pb by over 1 μg/dL unit (where $p < 0.001$). According to Table 3, residential street soil Pb is manifestly more important than other types of soil Pb in predicting variation in child blood Pb, although all soil Pb sources appear to increase child blood Pb greater than chance expectation. Overall, our suite of predictors accounts for about 76% of the between census-tract variation in child blood Pb.

Table 3
Random Effects Generalized Least Squares Regression Coefficients Predicting Blood Lead Levels in Children (μg/dL).

	Model 1 μg/dL	Model 2 μg/dL	Model 3 μg/dL	Model 4 μg/dL
Busy Street			0.279***	0.291***
			(0.102)	(0.096)
Foundation			0.401***	0.394***
			(0.088)	(0.083)
Open Space			0.185**	0.165*
			(0.093)	(0.087)
Residential Street			1.140***	1.098***
			(0.111)	(0.105)
Age		0.624***		0.623***
		(0.021)		(0.021)
Male		0.285***		0.302***
		(0.041)		(0.041)
Year	−0.094***	−0.116***		−0.111***
	(0.014)	(0.014)		(0.014)
Constant	5.739***	5.708***	5.521***	5.665***
	(0.115)	(0.109)	(0.062)	(0.069)
Wald X^2	46.05	1016.02	951.65	2082
R^2 between	0.256	0.336	0.754	0.769
N	55,551	53,348	54,052	51,905
Census tracts	280	280	272	272

Standard errors in parentheses *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

In Fig. 1, we show the marginal effects of soil Pb sources on child blood Pb through selected points in the sample space, while fixing control variables at their estimation sample means. On the *x*-axis, we plotted soil Pb quantities at various standard deviation values. On the *y*-axis, we plotted the marginal effect of children's blood Pb with respect to soil Pb source values. Our graphic shows the *percentage change* in child blood Pb for a percent increase from a set dosage of Pb content in neighborhood soils, corresponding to all four soil sample locations. Every *dose–response curve* corresponding to each soil Pb type is upward sloping, with the effect on child blood Pb increasing with soil Pb content. With regard to our main research question concerning which soil sample location exercises the greatest impact on child blood Pb, it is evident that residential street soil Pb sample locations are more important in predicting the upstream risk of child blood Pb.

Table 4 reports results from our decomposition of explained variance in Model 3 of Table 3, detailing the relative importance of each soil Pb type in explaining variations in child blood Pb levels. As a check on the calculation, note that the sum of derived *Soil Location Type Contribution* (C_i) values equals the observed R^2(0.754) in Model 3 of Table 3. Results show that a remarkable 39.07% of *Soil Location Type Contribution* in child blood Pb is attributable to residential street soil Pb, a value almost twice as high as observed for other soil Pb locations. The order of relative predictive importance of soil Pb location type is completed as: residential street 39.07%, busy street soil Pb (21.97%), open space soil Pb (20.25%) and home foundation soil Pb (18.71%).

Next, we derived the likelihoods of child blood Pb exceeding 5, 10, 15, and 20 μg/dL thresholds as a function of neighborhood soil Pb location types and relevant control variables. Table 5 and Fig. 2 report results for various random intercept logistic regression models. Once again, we found that residential street soil Pb location seems most important in determining whether a child eclipses a blood Pb threshold compared to other soil Pb location types. Other things held equal, a standard deviation increase in residential street soil Pb increases the odds of a child's blood Pb exceeding 5, 10, 15 and 20 μg/dL thresholds by multiplicative factors of 1.48, 1.71, 1.72, and 1.71, respectively. Whereas other soil Pb locations meaningfully impact the odds of child blood Pb exceeding measured thresholds, estimated effects are manifestly lower than observed for residential street soil Pb. Fig. 2 plots odds ratios corresponding to each soil Pb type reported in Table 5. Plotted point estimates are capped by 95% confidence intervals. It is noteworthy how the lower

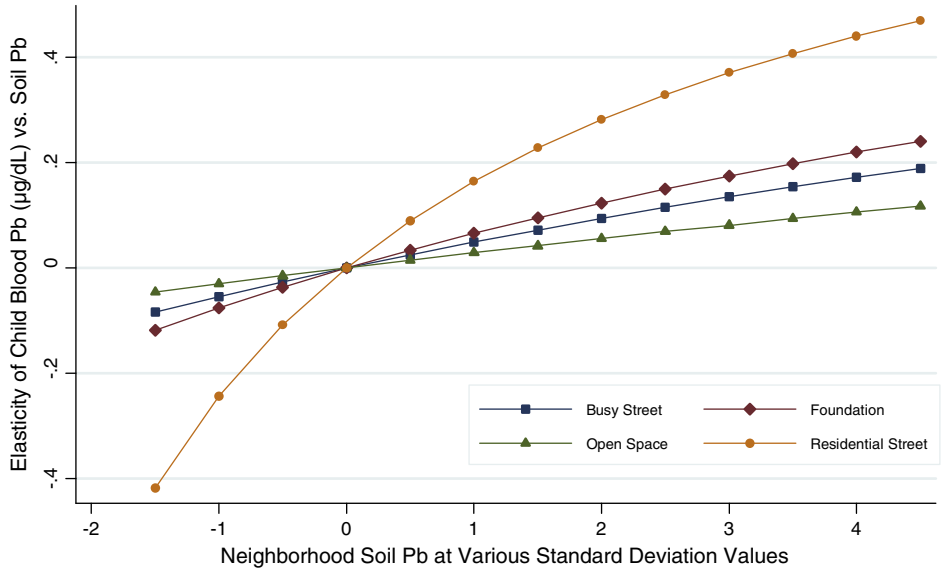

Fig. 1. Conditional elasticities* of child blood Pb (µg/dL) vs. community soil Pb by soil location. Note: *Marginal effects calculated on the basis of fully saturated Model 4 in Table 3, with control variables fixed at sample means.

estimate of risk from residential street soil locations exceeds or borders the upper estimate of all other soil Pb sample locations analyzed (Fig. 2).

Finally, to establish the robustness of our statistical observations, we completed our analysis with a series of quantile regressions to test whether our results on the predictive power of soil location sources of Pb behave similarly across parametric and non-parametric procedures. We modeled quantile points (τ) of 0.2, 0.4, 0.5, 0.6, and 0.8 in the conditional distribution of child blood Pb as a function of neighborhood soil Pb locations (Koenker and Hallock, 2001). Table 6 shows the non-parametric quantile regression results in which coefficients are expressed in standard deviation terms. As with our parametric models, results show that residential street soil Pb is the strongest predictor of child blood Pb across all quantile points. In fact, in Model 5, where $\tau = 0.8$, we find that the residential street soil Pb is an order of magnitude stronger ($b = 1.86$, $p < 0.001$) in predictive power than the next most powerful soil location source, busy streets ($b = 0.65$, $p < 0.001$). Overall, quantile regression results strongly corroborate results from parametric models reported in Tables 3 and 5.

4. Discussion

4.1. Blood Pb and environmental Pb discovery

With respect to the association between soil Pb and blood Pb, studies in Syracuse, New York; Minneapolis and St. Paul, Minnesota; Detroit, Michigan; and New Orleans, Louisiana have arrived at similar conclusions: One can predict spatial variation in children's blood Pb outcomes from the accumulation of Pb in neighborhood soils (Johnson and Bretsch, 2002; Laidlaw et al., 2005; Mielke et al., 1989, 1997, 1999, 2007; Zahran et al., 2011, 2013). Our study indicates that soils adjacent to residential and busy streets account for the bulk of between-neighborhood variation in child blood Pb levels. Whether the accumulation of Pb near roadways reflects the legacy Pb contamination from gasoline (Laidlaw et al., 2012; Zahran et al., 2013) or the routine demolition of buildings resulting in the release of lead-based paint (Farfel et al., 2003; Rabito et al., 2007), the high-energy environment present on roadways induces atmospheric re-suspension of Pb particles and increases inhalation and accidental ingestion risk facing children (Sabin et al., 2006; Sternbeck et al., 2002; Zahran et al., 2013).

Table 4
Calculation of soil source contribution (C_i) to $R^2_{0.1234}$.

	Order of squared semi-partial				C_i	% C_i
	Zero V_{ij}	First V_{ij}	Second V_{ij}	Third V_{ij}		
Busy Street	0.532	0.093	0.030	0.008	0.166	21.97%
Foundation	0.470	0.062	0.020	0.012	0.141	18.71%
Open Space	0.510	0.077	0.019	0.004	0.153	20.25%
Residential Street	0.719	0.231	0.134	0.094	0.295	39.07%

As compared to Model 3 in Table 3 (where all soil location types are included as regressors), a spare model including only residential and busy street soil samples results in very modest loss of predictive efficacy ($R^2_{O.sbfo} = 0.754$ versus $R^2_{O.sb} = 0.733$), indicating that one can adequately describe neighborhood soil Pb risk with significant reduction in the quantity of soil samples and by concentrating sampling efforts near roadways. In support of this statistical conclusion, Fig. 3 shows the geography of soil Pb in metropolitan New Orleans based only on residential street samples. As compared to the lead map of New Orleans based on all soil sample locations (see Mielke et al., 2011b), Fig. 3 closely approximates the known spatial variation of child risk of exposure to soil Pb.

As noted in the introduction, the medical model of environmental Pb discovery is a downstream methodology, wherein the search for environmental Pb follows from the detection of elevated Pb in a child's blood. By combining two sets of data, the statistical results of our study can meaningfully inform a *preventive model* of environmental Pb discovery at the neighborhood scale. Just as the age of housing stock in an urban area is used as a statistical shortcut for child risk of exposure to lead-based paint, our results indicate that one can shortcut the characterization of child risk of exposure to soil Pb by concentrating sampling efforts on soil adjacent to residential and busy streets, thereby significantly reducing the total costs (in time, money, and effort) of collection and laboratory work. If a neighborhood soil Pb risk is found to exist, then planning for intervention and additional sampling to characterize the source of exposure is warranted (Bugdalski et al., 2013). Indeed, the approach recommended here may be expedited by improvements in accuracy

Table 5
Random effects logistic regression odds ratios[†] (and 95% CI) predicting threshold exceedance of blood lead levels in children.

	Model 1 μg/dL > 5	Model 2 μg/dL > 10	Model 3 μg/dL > 15	Model 4 μg/dL > 20
Busy street	1.139 (1.037 to 1.250)	1.203 (1.048 to 1.381)	1.266 (1.089 to 1.473)	1.285 (1.078 to 1.532)
Foundation	1.194 (1.102 to 1.295)	1.313 (1.171 to 1.472)	1.270 (1.123 to 1.435)	1.323 (1.151 to 1.522)
Open space	1.140 (1.047 to 1.241)	1.169 (1.035 to 1.320)	1.188 (1.044 to 1.352)	1.138 (0.982 to 1.318)
Residential street	1.484 (1.340 to 1.644)	1.708 (1.478 to 1.975)	1.715 (1.468 to 2.003)	1.714 (1.434 to 2.049)
Age	1.379 (1.353 to 1.406)	1.372 (1.334 to 1.411)	1.323 (1.271 to 1.377)	1.257 (1.189 to 1.330)
Male	1.149 (1.106 to 1.194)	1.167 (1.104 to 1.234)	1.206 (1.114 to 1.307)	1.234 (1.102 to 1.381)
Year	0.975 (0.962 to 0.988)	0.907 (0.890 to 0.925)	0.923 (0.898 to 0.948)	0.948 (0.913 to 0.984)
Wald x^2	1807.10	1190.45	730.31	425.48
Log likelihood	−30,883.58	−16,634.33	−9367.41	−5386.77
N	51,905	51,905	51,905	51,905
census tracts	272	272	272	272

Note: [†]Confidence intervals in parentheses.

and reliability of hand-held x-ray fluorescence techniques (XRF), which permit soil metals to be measured directly in the field in a few minutes per site.

4.2. Limitations

While our results provide guidance on the relative importance of soil sample locations in predicting the upstream risk of child lead poisoning, our paper does not technically satisfy an economic definition of efficiency. With valid data on the marginal costs of soil sampling and analysis, and greater precision with respect to the declining marginal benefit (in terms of predictive accuracy) in the quantity of soil samples taken, one can determine the equilibrium quantity of soil samples necessary to optimize total benefits over total costs, and the optimal cost-effective sampling strategy across soil location types. Still, across parametric and non-parametric regression procedures, as well as results from our decomposition of between-neighborhood explained variance, we believe that a technically optimal sampling protocol for characterizing neighborhood-level risk of exposure to Pb contaminated soils is to likely involve a significantly lower number of samples than previously assumed and drawn primarily from residential and busy streets soils.

Additionally, while our study provides useful guidance on where to sample in an urban environment to maximize predictive efficiency, this study does not attempt to apportion sources and cannot be used to infer the source of Pb based on sample location. While it may be reasonable to assume that home foundation soils are more likely sourced by the deterioration and haphazard removal of exterior and interior lead-based paints, or that soils proximate to roadsides are more likely sourced by the legacy of leaded gasoline emissions, home and building demolition practices and local weather regimes complicate these simple distinctions. Efforts to identify the multi-media origins of environmental Pb can theoretically tune our statistically-derived guidance to more efficiently characterize neighborhood-level risk of soil Pb exposure but such exercises are beyond the scope of this work.

4.3. Soil guideline values and intervention

With respect to the risk of soil Pb exposure, the US EPA guideline values for soil Pb intervention are 400 ppm for bare soil where children play and 1200 ppm for surrounding soils (U.S. EPA, 2000). These guideline values for soil Pb exceed most international guideline values (Jennings, 2013). Moreover, remediating a single patch of private property abutting a residential street is unlikely to fully insulate

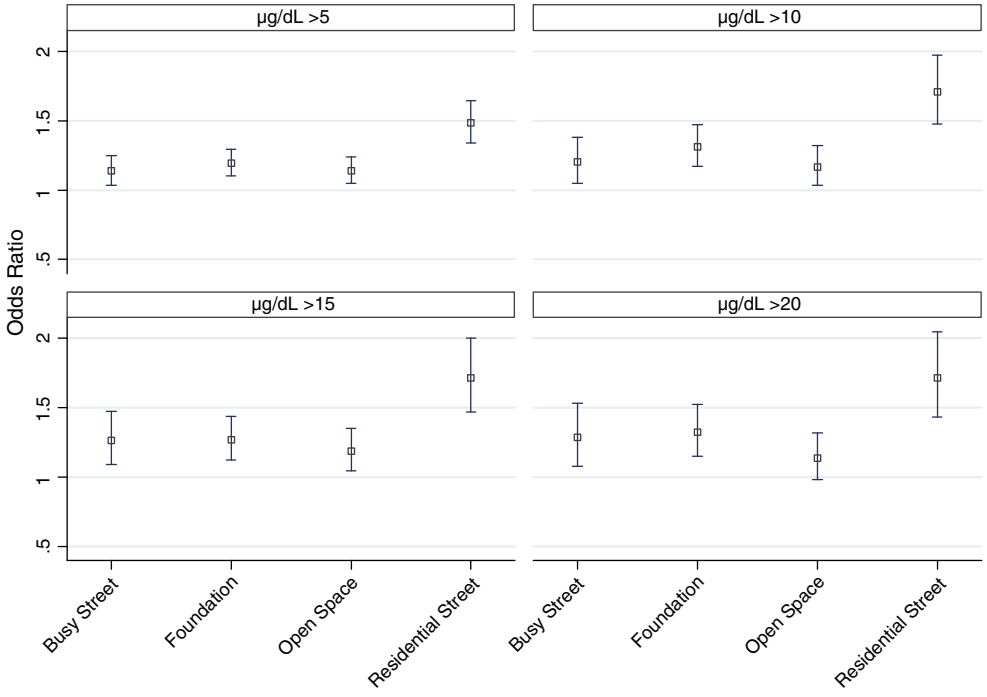

Fig. 2. Random effects logistic regression odds ratios (and 95% CI) predicting blood lead levels in children.

Table 6
Quantile regressions estimates predicting blood Pb LEVELS IN CHILDREN (μg/dL).

	Model 1 $\tau = 0.2$	Model 2 $\tau = 0.4$	Model 3 $\tau = 0.5$	Model 4 $\tau = 0.6$	Model 4 $\tau = 0.8$
Busy street	0.082***	0.202***	0.301***	0.382***	0.650***
	(0.017)	(0.015)	(0.016)	(0.024)	(0.047)
Foundation	0.097***	0.178***	0.233***	0.259***	0.475***
	(0.0143)	(0.013)	(0.014)	(0.021)	(0.041)
Open space	0.060***	0.083***	0.155***	0.172***	0.124***
	(0.015)	(0.014)	(0.015)	(0.022)	(0.045)
Residential street	0.313***	0.634***	0.765***	1.051***	1.864***
	(0.018)	(0.017)	(0.018)	(0.027)	(0.054)
Age	0.325***	0.248***	0.381***	0.513***	0.787***
	(0.010)	(0.009)	(0.009)	(0.013)	(0.025)
Male	0.068***	0.100***	0.149***	0.194***	0.290***
	(0.019)	(0.017)	(0.018)	(0.027)	(0.054)
Year	−0.048***	−0.047***	−0.064***	−0.075***	−0.138***
	(0.006)	(0.006)	(0.006)	(0.009)	(0.019)
Constant	2.882***	3.960***	4.593***	5.354***	7.979***
	(0.019)	(0.017)	(0.018)	(0.028)	(0.055)
Raw sum deviations	81,443.12	137,700.4	157,539.7	170,359	160,898.7
Min. sum deviations	79,244.56	127,793.4	143,209.5	151,574.7	136,845.2
N	51,905	51,905	51,905	51,905	51,905

Standard errors in parentheses.
*** $p < 0.01$.

a child from soil Pb exposure (Laidlaw et al., 2012), because Pb risk permeates residential neighborhoods. Results from extensive studies in three major US cities demonstrate the importance of mitigating multiple routes of exposures (Elias et al., 1996). For example, remediation of outdoor soil lead has very little benefit to children that lived in high-rise apartments with interior dust containing high levels of lead (Aschengrau et al., 1994).

International precedence for implementing a national clean soil program (including both inorganic and organic toxins) has been established by the Norwegian government (Ottesen et al., 2008). Given the widespread availability of low Pb (<20 mg/kg) soil concentrations in the U.S., intervention is possible anywhere (Gustavsson et al., 2001). New Orleans is positioned at the Mississippi River Delta which is composed of sediments containing only trace amounts of Pb transported by the River from the abundant and fertile soils of North American (Mielke et al., 2000). Inner city children, regardless of whether they live in public or private housing, have an incidence of Pb exposure of nearly 30% ≥5 μg/dL (Mielke et al., 2011b, 2013). As New Orleans rebuilds residential neighborhoods damaged by Hurricanes Katrina and Rita, there exists an opportunity to protect children into the future by applying a proactive, upstream public health policy to intervene on Pb and other toxins that have accumulated in the soils and play areas of the city. During the past century the growth of automobile oriented urban sprawl or peri-urbanization share similar processes of Pb dust emission and deposition as observed in New Orleans, and thus the results are probably applicable to all major cities.

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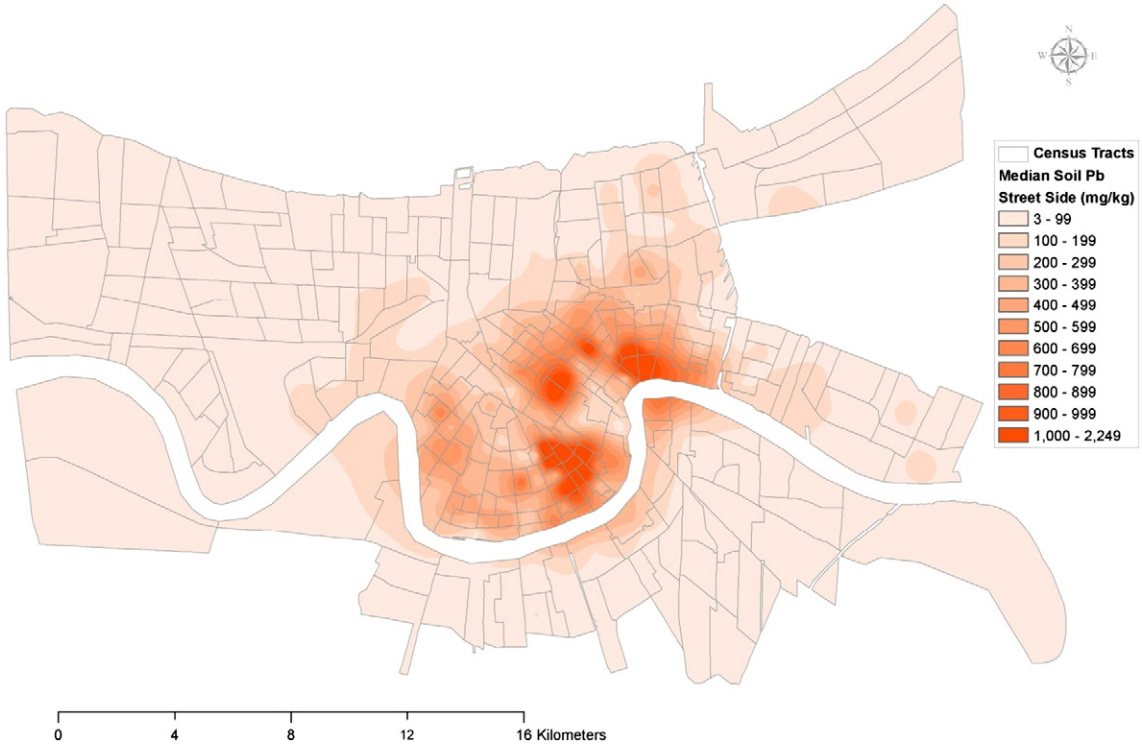

Fig. 3. Lead map of New Orleans based on residential street side samples.

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Chapter 4 - Paper 4: "*The Elephant in the Playground: Confronting Pb-contaminated soils as an important source of Pb burdens to urban populations*."

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The Elephant in the Playground

confronting lead-contaminated soils as an important source of lead burdens to urban populations

Gabriel M. Filippelli and Mark A. S. Laidlaw

ABSTRACT Although significant headway has been made over the past 50 years in understanding and reducing the sources and health risks of lead, the incidence of lead poisoning remains shockingly high in urban regions of the United States. At particular risk are poor people who inhabit the polluted centers of our older cities without the benefits of adequate nutrition, education, and access to health care. To provide a future with fewer environmental and health burdens related to lead, we need to consider the multiple pathways of lead exposure in children, including their continued contact with dust derived from inner-city soils. Recent research into the causes of seasonal variations in blood-lead levels among children has confirmed the importance of soil in lead exposure. "Capping" lead-contaminated soil with lead-free soil or soil amendment appears to be a simple and cost-effective way to reduce the lead load for urban youth.

THERE IS A COMMON—but misguided—perception that lead poisoning is no longer a public-health problem in the United States. Indeed, effective regulations against leaded gasoline and lead-based paint have dramatically reduced lead exposure. Unfortunately, however, the threat to urban neighborhoods across the nation is still very real.

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In the 20th century, two new applications turned lead toxicity into a widespread problem. First, lead-based paints became the gold standard for new homes in the early part of the century, prized for their durability and bright white color. Second, lead additives for gasoline were developed as an "anti-knock" engine formula in the 1920s, and the explosion of motor vehicles in the middle part of the century was fueled by gasoline doped with tetra-ethyl lead. By the 1970s, Americans encountered lead at every turn.

A number of scientific champions brought lead hazards to the public's attention. In the 1950s, Cal Tech geochemist Clair Patterson was conducting experiments to pinpoint the age of various rocks, but his results were skewed by consistent lead contamination. Further studies showed that lead levels were elevated in certain waters, soils, organisms (Settle and Patterson 1980), even Arctic ice—and most troubling, in the human body. Over the next three decades, Patterson helped to lead a crusade against lead that attracted the vociferous opposition of industry groups. But this effort eventually convinced lawmakers and regulators to outlaw lead in pipes, solder, and finally in gasoline (Bryson 2003). In a parallel fashion, Herbert Needleman fought against industrial, and even university, opponents to his findings of irreversible neurological defects as a result of lead poisoning of children (Needleman, Tuncay, and Shapiro 1972; for an account of this struggle, see Rosner and Markowitz 2005). As a result of efforts by Patterson and Needleman, among others, the number of children affected by lead poisoning in the United States has been reduced by over 80%. In the movies, this triumph would signal the closing credits—but in the real world, the story continues.

While less than 2% of children aged 0–5 years in the United States suffer from lead poisoning today (a value much improved but still a serious public health epidemic), children living in the urban centers of the East and Midwest have lead poisoning rates of 15–20% (NHANES 2003). In 1980, Clair Patterson presaged the current state of environmental insults to urban populations: "Sometime in the near future it probably will be shown that the older urban areas of the United States have been rendered more or less uninhabitable by the millions of tons of poisonous industrial lead residues that have accumulated in cities during the past century" (NRC 1980, p. 271). While many might consider lead poisoning a closed chapter in the annals of public health, recent research shows that the dangers still exist, and that they are elevated among the most at-risk children in our society.

In this essay, we discuss how children are exposed to lead and how they are affected, where the continued sources of lead are in urban areas, and how earth scientists can inform health scientists to enhance the health of the population—particular the poor people who typically inhabit the polluted centers of our older cities. These children face a number of challenges related to lead poisoning, including inadequate nutrition, which leads to soil pica behavior, as well as higher lead absorption rates due to iron deficiency anemia, and inadequate edu-

cation and access to health care. The primary cause of chronic lead loading in urban youth is their continued contact with dust derived from lead-enriched inner-city soils. This new paradigm of the exposure pathway of children to lead, which has been verified by our recent research into the causes of seasonal swings in children's blood lead levels, points to a relatively simple and cost-effective way toward reducing the lead load for urban youth.

EFFECTS OF LEAD ON HUMANS

Compared to other chemicals of environmental concern, the uptake mechanisms and toxicological effects of lead are relatively well understood. The major pathway of lead uptake in humans is via ingestion, where lead is absorbed in the intestine and incorporated in the body, initiating a series of toxicological effects. Physiological absorption potential for lead is dependent mainly on age: the portion of ingested lead that is taken up in the body is typically less than 5% for adults, whereas it is as high as 50% for children (Maddaloni et al. 1998; Ziegler et al. 1978). Because of their high absorption efficiency and the rapid neural differentiation during early brain and nervous system development, children are especially vulnerable to permanent effects of lead poisoning, which include irreversible neurologic disorders such as lowered intelligence and increased rates of attention-deficit hyperactivity disorder. When lead is incorporated into bone material, the bone becomes a long-term source of lead in the body, releasing that lead on time scales of months to years. For this reason, children treated by medical interventions like blood chelation may continue to have toxic levels of lead in their blood (Roberts et al. 2001). Furthermore, as neither the placenta nor mammary glands are a perfect barrier to lead, pregnant and lactating mothers with elevated blood-lead levels may themselves pose a health risk to babies and fetuses.

The health standards for lead levels in blood have been revised downward over the years as medical research has determined toxicological effects of lead in even low quantities. The U.S. Centers for Disease Control and Prevention (CDC) in 1991 chose 10 micrograms per deciliter as an initial screening level for lead in children's blood, although subsequent studies are still unable to find a "safe" lower level of lead, with levels below 10 micrograms per deciliter still causing some toxicological effects (Canfield et al. 2003; Chiodo et al. 2007; Nigg et al. 2008). The full spectrum of toxicological effects of lead is still not known and deserves further study. But the persistent presence of lead in children is a public-health issue of the first order.

From Occupational to Residential: The Paint Problem

Lead has been used by humans for thousands of years and its toxicity has been known for centuries, but it was not until the Industrial Revolution that this issue became a widespread problem. Lead was once a topic of concern mainly in occupational health circles, as severe lead poisoning was observed in industrial facilities including tetraethyl lead plants (Markowitz and Rosner 2002). But in the late 1800s, lead crept into homes in paint. Lead had been added to paint for centuries—distinctive colors are achieved with the addition of metals to paints, with bold white being the benefit of lead addition. However, a boom in residential housing development in the early part of the 20th century resulted in national-scale advertising blitz for "white lead paint" and the application of lead-based paints in millions of new homes. The addition of lead, in practice up to 15% by weight, enhanced the durability and flexibility of paints. Many single- and multi-family dwellings had lead-based paints in their walls, window sashes, and doorways. Even brick and stone houses often employed lead-based paints in windows and doors. Although lead enhanced durability, paint has its functional limits, and its degradation around friction points (doorways, window sashes), combined with the exploratory nature and oral fixation of young children, resulted in the first widespread tragedy of lead. As children were being admitted to hospitals with symptoms of severe and chronic lead poisoning, the link to lead-based paints became apparent. In the 1940s, pressure from the health profession and consumer advocate groups succeeded in legislation prohibiting the addition of lead to house paints. Although lead is still allowed in industrial applications like bridge paints, the banning of lead in house paints, which began in 1950 and became final in the early 1970s, gave hope for a lead-free future for children. This lead-free future never came to fruition for two reasons: the explosion of automobile use after World War II, fueled by leaded gasoline (more on this later), and the inevitable degradation of lead-based paint in and around homes.

The continued poisoning of children from lead-based paints was a sadly predictable outcome. The fact that house paint applied after 1950 was usually lead-free didn't change the lead content of old paint. Anybody who has refinished an older home is aware of the problem—what do you do with the lead paint on the walls, sills, and doorways? The popular way to refinish trim work and windows is the most problematic. Sanding of lead-based paints converts the paint from a glue-type solid with limited bioavailability into millions of fine particulates with relatively high lead content *and* very high bioavailability, due to the high surface-area/mass ratio of these particles. In many of the acute cases of lead poisoning in children in the United States today, contact with lead has resulted from home refinishing or remodeling. This problem can bridge class and race—rehabbing of older homes is often a luxury of the upper-middle class, as they restore a historic home to its original luster.

To confront this problem, many health and environmental agencies at the national, state, and local levels have been waging a war of remediation and education about the hazards of lead. Most of the remediation efforts have been focused on safely removing or covering lead-based paints in homes. With millions of dollars in grants and incentives to owners and landlords, lead-based paints have been removed or covered all over the country. The agencies involved have touted these efforts as a success, holding up the clear improvement in the number of children affected by lead over the past 25 years. In a national health assessment survey in the late 1970s, 88% of the nation's children (0–5 years of age) had blood-lead levels above that deemed safe by today's standards (10 micrograms per deciliter); in a follow-up survey in the 1990s, that number was down to 2.2% (NHANES 2003), with annual improvements seen in interim surveys up to today. When people are asked what they consider the key pathway for lead to children, they invariably respond that kids get lead poisoning from eating paint chips.

When medical, scientific, and regulatory findings reach the collective psyche of society, a paradigm is formed. This paradigm, that lead-based paints still constitute the biggest risk to children with respect to lead, and that the remediation of lead-based paint sources has in the past and will continue in the future to provide the chief benefits to children's health, is firmly entrenched. The seduction of this idea is easy to see—images of toddler's bite marks of painted window sills, X-rays revealing paint chips in a child's stomach, a photo of a white-clothed team of remediation experts removing lead-based paint from a building—can be superimposed to create an image of a neat, clean, and effective solution to this problem. This seduction is now even in the courtroom, where several high-profile cases brought before juries revolve around large paint producers, like Sherwin-Williams, who are being sued for producing lead-based paints over 60 years ago. Clearly, corporate and industrial responsibility should extend to producing products that knowingly endanger the health of people and the environment, and the idea of reparations to support remediation of this public-health menace is appealing.

But what if the paradigm is inadequate? What if poor, urban youth are no longer being poisoned just by chipping paint, but now predominantly by the soil around them? What if the vision of a white-clad team of specialists sweeping through a housing project, removing lead from the walls and leaving in its wake sparkling new lead-free paint, needs to be replaced by an image much more messy and comprehensive to solve this problem of social injustice? How will we know when to shift resources for education and remediation in another direction? We believe that the time is now. This belief is bolstered by a series of findings that suggest that soil—particularly the fine dust that derives from soil during dry periods and blankets horizontal surfaces inside and outside of homes—is a prime culprit in the poisoning of our children by lead. The inability of remediation of paint alone to reduce the blood-lead levels of urban youth is one clue that we have been missing a key additional source of lead to our children.

Lead-Contaminated Dust from Soils as a Vehicle for Chronic Lead Poisoning of Urban Youth

We have hit the wall in terms of improving the lead-poisoning outlook for some children, particular those living at or below the poverty level in older cities. Even after decades of active intervention, these urban youth have lead-poisoning rates that are up to 10 times the national average. In 1994, a summary statement from a national health survey stated that "the exposure to lead at levels that may adversely affect the health of children remains a problem especially for those who are minority, urban, and from low-income families. Strategies to identify the most vulnerable risk groups are necessary to further reduce lead exposure in the United States" (Brody et al. 1994, p. 277). These socioeconomic risk factors include poor nutrition with the potential for pica behavior (a subconscious desire to ingest soil and dust to overcome nutritional deficits), and inadequate pediatric health care. Additionally, and of critical importance for improving the health outcome of urban youth, these risk factors also include poor home maintenance with high rental percentages, significant proportions of urban housing with high dust and dirt exposure, and relatively low awareness of the links between behavior and health.

In particular, the continued poisoning of urban youth from the very dirt and soil upon which they live is the key to a new emerging paradigm—namely, that the continuing source of lead exposure to children is lead-enriched soils, and particularly dust resuspended from these soils, that are prevalent in cities, especially older ones (Filippelli et al. 2005; Laidlaw et al. 2005; Mielke and Reagan 1998). The source of lead to the soils includes degraded lead-based paints, but also lead deposited from tailpipes, the result of 60 years of leaded gasoline combustion. In fact, the improvement in the national average of blood-lead levels may be due in large part to the banning of lead as an additive in gasoline in 1980.

The production and use of lead for gasoline additives was spurred by the need to control the explosion of gasoline in the cylinders of internal combustion engines. The formulation of tetraethyl lead as a fuel additive was "perfected" in the 1920s, resulting in the adoption of a global fuel standard that contained about 2% lead oxide by weight. An early warning sign went up when scores of workers in plants producing tetra ethyl lead additives were severely poisoned by lead toxicity, although a concerted industrial cover-up limited public awareness of this situation (Markowitz and Rosner 2002). But concerns about the environmental impacts of tetraethyl lead were shelved as the automobile age dawned and affordable transportation dramatically altered the American landscape. The peak in lead use for this application followed the trend in automobile use in America: by 1970, 250,000 tons of lead were used in gasoline and emitted from tailpipes every year.

Roadway Sources of Lead

Overall, about 5 million metric tons of lead was deposited in the environment as a result of the combustion of leaded gasoline (Mielke et al. 1997). Almost all of that lead was originally deposited very close to roadways, with aerosolized combustion products containing lead initially deposited within about 50 meters of a roadway if no obstructions were present. The fate of deposited lead then depended on the conditions of the depositional area. Although intersections of busy streets may have received over one metric ton of lead per year, their impervious surfaces led to continual runoff of lead-enriched particulates down storm drains (and from there into treatment plants or directly into rivers). If the particulate lead was deposited instead on a grassy fringe, like a front yard or park, the lead was effectively retained (Filippelli et al. 2005). In such a setting, the insolubility of lead leads to surface peaks in lead concentration of soils; in relatively undisturbed soils, this surface-lead enrichment may be the product of decades of lead deposition and may reach levels above 1,000 ppm. Thus, surface soils became the repositories of lead deposited over decades—in the case of older roadways, the proximal soils might retain almost all of the lead deposited on them over a period of about 60 years.

The roadway lead is generally highly bioavailable. Immediately upon combustion, tetraethyl-derived lead is precipitated as tiny and poorly mineralized oxides and oxyhydroxides, both of which are much more susceptible to dissolution in gastric systems than is the well-mineralized lead found naturally in soils. Therefore, dust originating from urban soils contaminated by anthropogenic lead is more toxic per unit mass than naturally occurring lead dust. Because of its deposition source, much of the tetraethyl-derived lead is associated with clay size fraction (less than 0.2 microns in diameter) in urban soils. The small size leads to this clay soil fraction being the predominant component of soil "resuspended" during dry and windy periods. Thus, lead in dust blown from urban soils is more potent and concentrated than would be expected from simple measurements of the lead content of the bulk soil.

Diffuse Soil Lead and Children's Health

The original sources of lead in the environment were point sources, including lead-based paints, gasoline-emitted lead, and lead emitted from smelters. But an analysis of many urban areas reveals that these point sources have, to some extent at least, been redistributed to produce regions of lead enrichment (Filippelli et al. 2005). Several factors can lead to redistribution of lead-enriched particles and soil, but the recurrence of a general urban enrichment of soil lead, termed "diffuse soil lead," has been documented in many regions.

One of the characteristics of lead distribution in surface soils of several older cities is a distinct decreasing trend from city center to suburban surroundings, a legacy both of lead deposition, redistribution, and smearing of original point

sources, and less lead deposition in newer suburban neighborhoods due to recent lead controls. The urban roadway example shows both the impact of the point source of lead deposition from leaded gasoline as well as the diffuse soil lead that blankets urban regions. In other words, even at distances away from the roadway, beyond where direct lead deposition occurs (and far away from structures using leaded paint), the background level for lead is significantly higher in the urban roadway transects than in suburban transects. This urban-suburban gradient is one overriding factor affecting the amount of lead loading to individuals, a factor that we will discuss on a larger scale and with respect to human health.

In many urban areas of older cities, large segments of children below the age of six are above the action level for lead in blood; this has been well documented by Howard Mielke of Tulane University for New Orleans and Baltimore, David Johnson for Syracuse, and more recently by us for Indianapolis. The actual distribution of blood-lead levels exceeding action limits is getting more difficult to obtain due to privacy concerns, but in the past blood-lead values could be collected from health department records down to the level of a street address, providing an outstanding way to examine the environmental factors in human health.

To explore the concept of diffuse soil lead and its potential role in affecting children's health in Indianapolis, Filippelli et al. (2005) performed a coupled soil survey and epidemiological analysis. The soil sampling criteria included being greater than 50 meters from roadways and from structures that might have contributed lead-based paint, and was augmented by aerial photographic records over Indianapolis from several time slices (1940, 1970), to rule out the potential for inadvertently sampling soils from disturbed, excavated, or filled areas that might have surface-lead contents characteristic of artificial materials rather than natural soil. As one can imagine in a rapidly developing urban area, this criteria narrowed down acceptable sites to only about 100 distinct sites, many of which were in parks, cemeteries, and school grounds. In contrast to roadway and house-side soil sampling, which might exhibit lead concentrations above 1,000 ppm, the highest soil-lead concentrations were below 480 ppm. The lowest lead concentrations averaged about 50 ppm, which is a typical value for soils in this region. The highest soil-lead concentrations were focused in a bulls-eye pattern directly over the old urban areas of Indianapolis, where the diffuse soil-lead content averaged over 200 ppm (Figure 1). Beyond this central hot spot, lead concentrations decreased toward the outskirts of the city, ultimately falling to background values in the suburban to rural fringes of the city. The central peak is consistent with the long history of lead use in the downtown, but the generally high values even away from point sources supports the argument of a redistribution of lead over time.

Combining the distribution of soil lead with that of children's blood-lead poisoning reveals several important characteristics of diffuse soil lead as a potential contributor to children's health. First, the similarity in the distribution of ele-

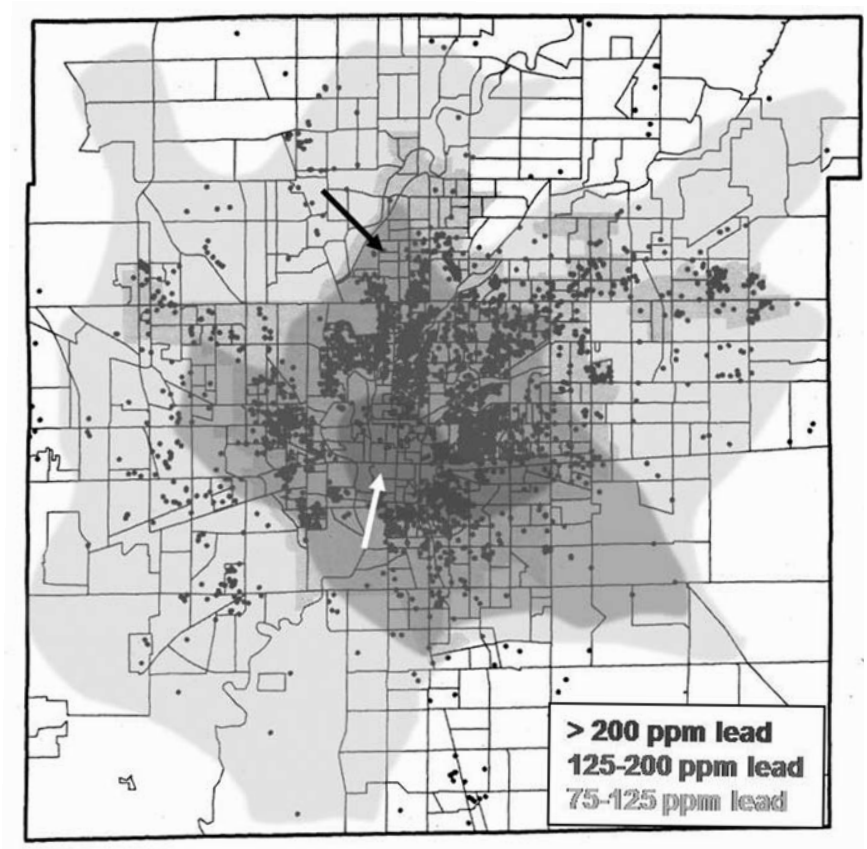

FIGURE 1

Satellite infrared image of Indianapolis (Marion County) in central Indiana; north is up. The concentration of diffuse soil lead (in shaded regions) displays a characteristic pattern of urban enrichment trending toward background values in suburban and rural areas. The overprint of high diffuse soil lead presented here corresponds roughly with the distribution of elevated blood-lead levels in children, displayed as circles for the distribution of children's venous blood samples exhibiting lead concentrations above the level of concern (10 micrograms per deciliter) from 1992 to 1994 in Indianapolis (data from the Indiana State Department of Health). Most elevated blood samples are from the downtown region (significant overlap of multiple positive results occur in this region), with some additional scattered positive results ranging toward the older suburban development to the west and the east. The arrows point to regions with high soil lead but low incidence of lead poisoning, at apparent odds with the direct link between soils and blood. A number of factors, however—socioeconomic status, age, population distribution—act as filters between potential exposure and toxicology. In the case of the lighter lower arrow, the lack of lead poisoning is due to the lack of homes in this industrial corridor, and in the case of the darker upper arrow, this highlights a main street that displays a socioeconomic divide between poverty-line neighborhoods in the near-urban area and upper-middle and upper-class neighborhoods in the northern suburban area.

SOURCE: FILIPPELLI ET AL. 2005.

vated soil- and blood-lead values in the downtown areas reveals the potential for diffuse soil lead to be an additional and important factor in children's blood-lead levels. Second, population patterns definitely have some influence on the health distribution data. For example, some areas of the downtown have the highest concentration of diffuse soil lead but almost no incidences of childhood lead poisoning; in this case, this is because this region is an industrial area with no housing (Figure 1, lower lighter arrow). Finally, a socioeconomic filter likely comes between the exposure factor (diffuse soil lead) and the epidemiological factor (blood-lead levels). As an example, the central north-south thoroughfare in Indianapolis is called Meridian Street, which in its near-urban stretch is lined with apartment buildings and relatively low-income rental neighborhoods. This area exhibits high diffuse soil lead concentrations and a high incidence of blood-lead poisoning. But as Meridian moves north into the older suburbs, it becomes a National Historic Landmark, lined by mansions, including the Governor's Residence, with four-acre expansive manicured lawns. The streets bordering Meridian along this stretch are also characterized by well-maintained older homes, owned by upper-middle class families. Although diffuse soil lead concentrations are still high in this area, children's blood-lead levels are generally low; in fact, no incidences of blood-lead levels exceeding action limits were reported in 1992–1994 along this portion of Meridian and surrounding blocks (Figure 1, upper darker arrow).

Although many factors influence the relationship between geology and human health in the story of lead, it is clear that we do not yet understand all of the confounding factors. Furthermore, the generalized approach presented above provides a reference point for further work, but it does not effectively integrate health and geologic data, nor does it present clear recommendations that geologists can make to health specialists in further reducing this public-health hazard, beyond the incredibly costly and disruptive solution of removing all of the contaminated surface soil in urban areas and replacing with clean fill. Several bridging efforts are now being pursued to help further the human health–environmental quality linkage in the context of eliminating childhood lead poisoning. Beyond simply documenting lead distribution and its public-health implications, research has also examined lead in a more detailed manner as a toxicological agent with predictable behavior. For example, isotopic techniques have been utilized to examine the entry mechanisms of lead into the body and the cycling of lead within the body, with a goal of pinpointing lead toxicity in individuals and thus more closely coupling prevention and treatment (Graziano et al. 1996; Gwiazda and Smith 2000). Another tool of promise in assessing lead poisoning is predictive modeling of children's blood-lead levels using climatologic data.

Climatic Factors and a Blood-Lead Predictive Model for Health Care

Several studies have identified a seasonal trend in blood-lead levels, with average monthly blood-lead levels of children from urban areas increasing significantly in summer months, perhaps partly due to increased exposure to lead-based paint on window sills and through increased contact with soils containing lead during the summer. Summer increases of children's blood-lead levels were so prominent over many years in Syracuse that a group researchers led by David Johnson at the State University of New York, College of Environmental Science and Forestry, concluded that the phenomenon is probably caused by the interaction between climate and soils, leading to enhanced dust-lead loading to children (Johnson, McDade, and Griffith 1996).

To better understand this climate/soil/human health link, several projects are underway to investigate in detail variations in children's blood-lead levels as a function of climate and soil factors in several urban areas. The ultimate goal of this effort is to develop a predictive model, whereby a medical researcher can better assess the likelihood of lead poisoning based on seasonal and weather-related factors, as well as blood-lead level data. Laidlaw et al. (2005) used a number of independent climatologic variables, including average monthly soil moisture, PM10 (fine particulates less than 10 microns in diameter, an indicator of air quality and dust concentration in the atmosphere), wind speed, and temperature obtained from state and federal government data sources. They also used blood-lead databases obtained from local and state governmental sources.

This model indicates that soil moisture, wind speed, PM10, temperature, and the monthly dummy variables for March, April, June, July, August, and September explain 87% of the variation in monthly average child blood-lead level concentrations (Laidlaw et al. 2005). Based on this multiple regression model and recently published results from several other American cities (Laidlaw and Filippelli 2008), it appears that the seasonality in children's blood-lead levels is controlled by exposure to lead dust originating from contaminated soils and suspended in the air when several weather-related environmental conditions are present: temperature is high, soil moisture is low, and atmospheric PM10 is elevated (Figure 2). When temperature is high and evapotranspiration maximized, soil moisture becomes low, lead-enriched PM10 dust disperses in the urban environment and is manifest by elevated lead-dust loading. In this case, exposure is via increased dust loads in homes and on contact surfaces, with ingestion being the uptake mechanism and toddlers at greatest risk due to their behavior (crawling, tactile exploration, hand-to-mouth behavior). Although further work using detailed tracking of lead, possibly involving lead isotopic studies, may help to elucidate the connection between seasonality and blood-lead values, the ability of geochemical and meteorological factors to predict blood lead supports the supposition that external loading and exposure drives much of the blood-lead

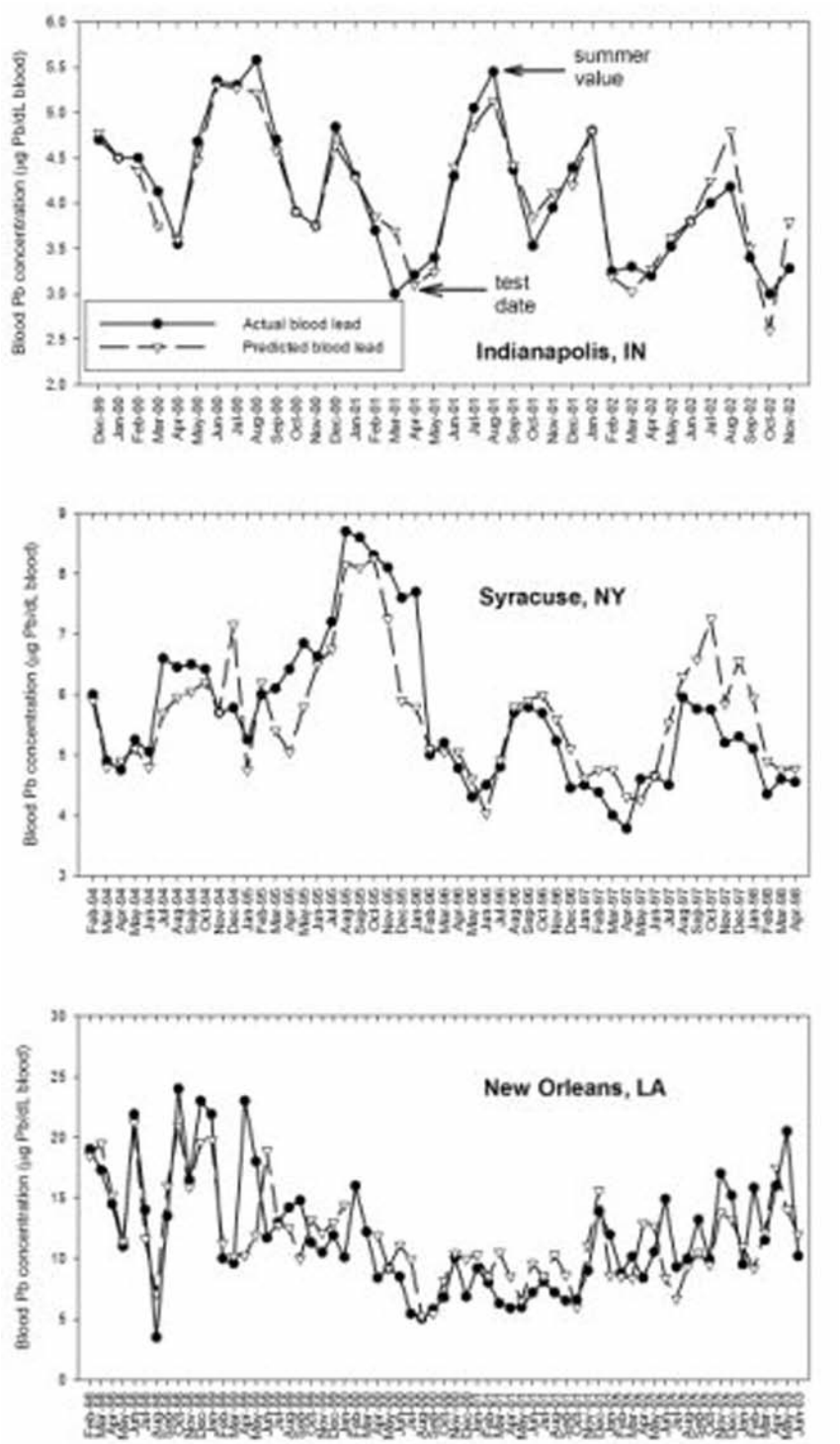
summer value
test date
Actual blood lead
Predicted blood lead
Indianapolis, IN
Blood Pb concentration (µg Pb/dL blood)
Syracuse, NY
New Orleans, LA

FIGURE 2

concentrations. Because resuspension of lead from contaminated soil appears to be driving seasonal child blood-lead fluctuations, lead contaminated soil in and of itself may be the primary driving mechanism of child blood-lead poisoning in the urban environment.

However, the seasonal deposition of lead-enriched dust and its ingestion by children may not be the only factor driving the observed seasonal patterns in blood-lead levels. Recent work indicates a potential role for increased sunlight-induced vitamin D synthesis in the summer, which increases gastrointestinal lead absorption and skeletal lead mobilization at least in children from 4–8 years of age (Kemp et al. 2007). In fact, multiple interacting causes may be at play, with factors including lead-enriched soil deposition, as well as age, race, sunlight exposure, and diet.

In addition to the development of hypotheses related to the incorporation of lead into children's system, a promising result of these modelling analyses is the ability to predict toxicity in a given population. In other words, through easily collected atmospheric and soil data a health researcher can determine the expected variation in blood-lead levels of the general population and use this data as a context for evaluating blood-lead level data from individual patients (Figure 2). This is particularly important when attempting to treat blood-lead poisoning using discrete venous sampling events: a blood-lead level for a given patient in the spring, under conditions of high soil moisture, could be significantly higher in the same patient just several months later, when atmospheric conditions increase ambient lead loading.

A NEW WAY FORWARD?

In summary, a newer paradigm of urban lead loading has emerged, one that helps to explain continued chronic lead poisoning and seasonal patterns in blood-lead levels of children. Unlike discrete point sources like lead paint and industrial contact, which are still responsible for most cases of acute lead poisoning, diffuse soil lead is the main avenue for urban lead loading of children. The diffuse soil lead comes from several sources, including leaded gasoline and degraded lead-based

FIGURE 2 CAPTION: *Best-fit model results to predict blood-lead levels in children from Indianapolis, Syracuse, and New Orleans, compared to actual monthly average blood-lead levels. This type of effort can be used to better treat lead poisoning from a public-health perspective by providing clinicians with predicted trends of blood-lead levels (functionally calculated as a percent deviation from mean) at a given blood sampling event, allowing them to calculate the potential increase or decrease with time given normal exposure. The clinician who is analyzing blood-lead test results from, for example, late winter–early spring, could predict that the patient's summertime blood-lead levels would likely be about 50% higher. If this higher predicted level is above the level of concern for the clinician, a follow-up test in the summer might be recommended.*

SOURCE: AFTER LAIDLAW ET AL. 2005.

paints, but in a sense the source no longer matters: because of the ability of surface soils to retain lead, these soils themselves have become the new risk factor for children's health in lead-loaded cities. If the action level for blood lead in children is dropped to 5 or even 2 micrograms per deciliter, we suggest that the dust resuspension paradigm will be central to predicting patterns in lower-level lead poisoning in children, leading to the need for new mitigation strategies.

Widespread contamination of urban soils creates a different challenge for mitigation of lead risks for children, one based on removing surface soils from human contact. Most mitigation efforts for heavily contaminated soils have involved soil removal and replacement, an extremely disruptive, expensive, and not terribly effective option for controlling lead sources in urban areas (Farrell et al. 1998; Weitzman et al. 1993). Recently, another approach, which is much cheaper and appears to be as effective as soil removal, was tested by Howard Mielke in New Orleans. Mielke's approach is simply to cover the contaminated yard soils with about 15 cm of lead-free soil, which in the case of New Orleans came from the nearby Mississippi levee (Mielke et al. 2006). At a fraction of the soil removal cost, this clean soil is simply graded over the old soil layer, hydroseeded (a slurry of seeds and moisture-retaining fill mixture sprayed onto the ground), and left to grow a lawn. This approach "caps" the lead-contaminated soils, removing them from contact by children. The result of initial work is a substantial reduction in the blood-lead levels of children living in the affected homes. Interestingly, Mielke observed that over the course of several months after treatment, soil lead levels in the treated sites began increasing. This increase was due to the resuspension and deposition of soil dust from adjacent untreated yards and neighborhoods that still had high soil lead concentrations. This finding not only supports the paradigm of diffuse soil lead as a culprit in urban areas, but it also indicates that a comprehensive treatment approach is required to provide a long-term benefit.

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CHAPTER 5

Discussion

5.1 DISCUSSION

The need to understand the major source of Pb in urban areas (UAs) is critical because there is an ongoing and unresolved epidemic of elevated children's PbB levels in many older urban metropolitan areas of the USA. By contrast, because of the lack of co-ordinated or systematic assessment the same evaluation is not possible for Australia, but the evidence indicates that the same problem as exists in the USA is also occurring in Australia. In many urban inner-city areas in the United States there is an epidemic of childhood PbB poisoning (Gould, 2009). It has been estimated that 24.5 %, or 9.6 million US children have a PbB in the 2–10 µg/dL range, a level which will cause sub-clinical signs (Gould, 2009). The United States Centers for Disease Control and Prevention (CDC) estimates that in the United States approximately 535,000 children aged 1–5 years had PbBs ≥5 µg/dL (CDC, 2013). These exposures are quite variable geographically with some locations more significantly affected. For example, in New Orleans, children currently have a PbB prevalence (> 5 µg/dL) of 29.6% (Mielke et al., 2013) and Detroit children (aged 0-10 years) have a PbB prevalence of 33% (> 5 µg/dL) (Zahran et al., 2013a). In 2012, the United States CDC Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP, 2012) recommended the adoption of a children's PbB reference level of 5 µg/dL.

Unlike the USA, the PbB prevalence in Australian UAs is unknown as Australia's government does not measure children's PbB systematically. However, Taylor, Winder and Lanphear (2012) estimate as many as 100,000 children may have a PbB > 5 µg/dL, which would not be unsurprising given that soil Pb concentrations in Sydney (Birch et al., 2011) are similar to those in New Orleans (Mielke, 1994; Mielke et al., 2005a). The research presented below attempts to elucidate the source and pathways of Pb in soil, the atmosphere and homes

and its human health risk in Australia and the United States. This contribution from this thesis study to the literature is also considered.

5.1.1 Estimation of Historical Pb Emissions in US Urban Areas

The *Environment International* (Mielke et al., 2011; Chapter 3) and *Science of the Total Environment* paper projects (Mielke et al., 2011; Chapter 3) have quantified the enormous amount of Pb that has been emitted into the atmosphere and deposited onto soils in UAs of California and the United States. Pb was used in equal amounts in both paint and petrol in the US in the 20th century - about 5 to 6 million metric tons each (Mielke and Reagan 1998). This is important because it establishes that petrol-derived Pb contributes greatly to the widespread contamination of urban soils in the United States. Prior to these papers, there were no estimates of Pb emissions in US cities. While no similar study has been performed in Australia, it is suggested that urban soils have been contaminated in a similar fashion. Two national assessments of petrol Pb emissions determined that in 1976, 3,842 tonnes of Pb were emitted in Australian capital cities and 2,388 tonnes of Pb were emitted in 1985 (Farrington and Boyd, 1976; Farrington, 1985).

5.1.2 Atmospheric Soil and Atmospheric Pb Seasonality

One of the objectives of the *Atmospheric Environment* (Laidlaw et al., 2012) study was to analyse temporal variations in atmospheric soil and Pb aerosols in four US cities: Pittsburgh, Pennsylvania Detroit, Michigan Chicago, Illinois and Birmingham, Alabama. The specific goals of the study were to test whether re-suspended urban soil was the dominant source of Pb aerosols in the four cities, and whether atmospheric soil and Pb aerosols follow seasonal patterns and if the highest concentrations occurred during the summer and/or autumn. Based on the results in the Laidlaw et al. (2012) study (Figure 5.1; Chapter 4), it has been

established that in four major American cities contemporary atmospheric soil becomes re-suspended in the summer and autumn and correlates with atmospheric Pb. This is a new finding as no studies are known internationally that quantify the temporal relationship between atmospheric soil and atmospheric Pb using high quality datasets like those used from the USEPA IMPROVE monitoring station. Another new finding from the Laidlaw et al., (2012) project is that atmospheric soil and atmospheric Pb levels were observed to be approximately three times higher during weekdays than weekends. We interpreted these data as suggesting that the dominant process of re-suspension of urban soil is being controlled by traffic turbulence re-suspending highly contaminated roadside soils (Laidlaw and Filippelli, 2008). These findings are important because they indicate that contemporary urban atmospheric Pb is associated with a contaminated roadside soil source and suggests that any attempts to reduce urban atmospheric Pb must involve remediation of soils, especially roadside soils. The re-suspension of Pb contaminated roadside soils is also concerning due to the large number of schools and child care centres located on main roads in Sydney. No other studies internationally have observed similar findings or concluded that re-suspension of roadside soils may be causing PbB seasonality patterns in children.

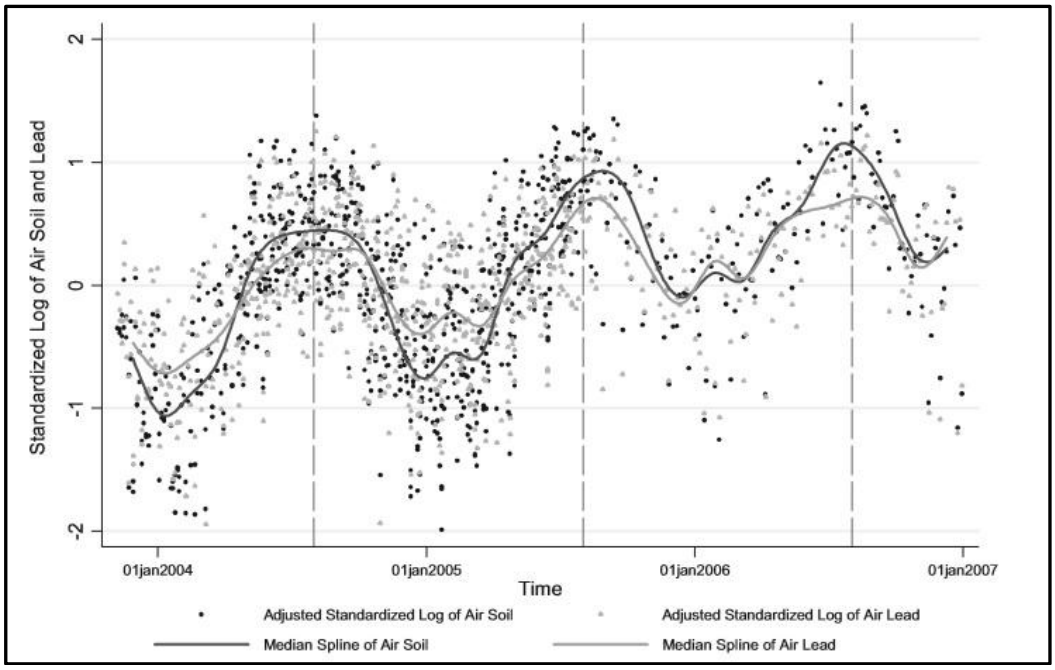

Figure 5.1 - Weather adjusted air Pb and air soil over time, including median spline fits, for Pittsburgh, Detroit, Chicago and Birmingham (from Laidlaw et al., 2012).

The re-suspension findings from the studies in this thesis (Chapter 4) are consistent with previous findings presented in the research literature. Limited data has been published on the seasonal variations in atmospheric Pb in the United States. Summer and autumn maxima of atmospheric Pb have been observed in Washington D.C. (Green and Morris, 2006), (Melaku et al. 2008)), Boston (USEPA, 1995), New York (Billick et al., 1979), and Chicago (Paode et al., 1998). In Boston (USEPA, 1995), modelled Pb levels for air, floor dust and furniture dust all had maxima in July. In Jersey City, New Jersey, Yiin et al. (2000) observed that windowsill wipe samples were most correlated with PbB concentration and the variation in dust Pb levels for floor Pb loading, windowsill Pb loading, and carpet Pb concentration were consistent with the variation of PbB levels. The highest levels occurred in the hottest months of the year (June, July, and August). In New Jersey (Edwards et al., 1998) found that the mean summertime household dust loadings were 68% higher than mean winter household dust loadings. Edwards et al. (1998) also observed that the dust mass deposition rate in summer (0.37 ± 0.13 μg/cm^2/day) was almost twice as great as winter (0.22 ± 0.13

µg/cm^2/day). In Mexico City, Rosas et al. (1995) observed that during rainy seasons of the year, PM_{10} dust was settled and atmospheric Pb concentrations were lower; during seasons with low rainfall PM_{10} and atmospheric Pb concentrations increased. Interior summertime maxima Pb loading rates were also observed inside a home in Northern England (Al-Radady et al., 1994). Al Radady et al. (1994) observed that between April and July (spring- summer), dust Pb concentrations increased on the walls (from 0.49 to 0.89 µg/m^2 per day), furniture (from 1.84 to 2.41 µg/m^2 per day), curtains (from 2.55 to 4.45 µg/m^2 per day), and window sills (from 2.57 to 5.86 µg/m^2 per day).

The literature (e.g. Chapter 1) supports the contention that roadside soil re-suspension is a major source of Pb poisoning children. It is becoming better understood that the process involves re-suspension of highly bio-available and Pb contaminated roadside soils due to turbulence caused by trucks and automobiles on high traffic roadways (Cho et al., 2011, Laidlaw et al., 2012). The soil beneath grass lawns is not static. Sutherland and Tolosa (2001) indicate that soil sediment is discharged (remobilised) at the edge of the interface between grass and street, especially after rains, and when the lawn is higher than the curb side. The soil particulates become a source of metals on streets, where they can be re-suspended after the soil dries (Sutherland and Tolosa, 2001). In Sydney, Australia Davis and Birch (2011) measured Pb loading rates beside roadways with varying traffic rates and observed that Pb loading rates were highest beside high traffic roadways (see Table 5.1). This study was conducted between 2007 and 2008, approximately 5 years following the elimination of Pb in petrol in Australia. In Berlin, Germany, Lenschow et al. (2001) observed that at curb-sides on main streets, the PM_{10} concentration is up to 40% higher than the urban background with half of this additional pollution due to motor vehicle exhaust emission and tire abrasion and the other half due to re-suspended soil particles. Soil re-suspension has been observed to be

higher in the older UAs with more traffic than the newer suburban areas. Simons et al. (2007) observed significant differences in particulate loading between urban and suburban areas in Baltimore, Maryland, with urban PM_{10} concentrations of 47 $\mu g/m^3$ versus 8.7 $\mu g/m^3$ in suburban areas, and urban $PM_{2.5}$ concentrations of 34 $\mu g/m^3$ versus 18 $\mu g/m^3$ in suburban areas. Since PM_{10} often consists of large portions of soil dust particles, these data suggests that urban atmospheric soil loading rates are significantly greater than suburban soil loading rates, irrespective of seasonal differences.

	Vehicles/Day	**Total Particulate**	**Pb**
		(mg/m^2 day^{-1})	($\mu g/m^2$ day^{-1})
Background-L	NA	19	12
Background-H	NA	30	29
Road-L	2000	58	38
Road-Ma	47500	212	83
Road-H	84500	288	106

Table 5.1 – Pb and Total Particulate Loading Rates measured beside roadways in Sydney, Australia between 2007 and 2008 when Pb was no longer used in petrol (Davis and Birch, 2011).

5.1.3 Association between Atmospheric Soil, Atmospheric Pb, and Children's Blood Pb levels

Compared to the reference month of January, child PbB levels in Detroit are found to be between 11% and 14% higher in the months of July, August, and September. Explaining this seasonal phenomenon was the aim of our *Environmental Science and Technology* study (Zahran et al., 2013a; Chapter 4). This study evaluated atmospheric concentrations of soil and Pb aerosols, and PbB levels in 367,839 children (ages 0-10) in Detroit, Michigan USA from 2001 to 2009 in order to test a hypothesized soil → air dust → child pathway of contemporary Pb risk (Zahran et al., 2013a). This study establishes firmly that children's PbB levels are

strongly associated with atmospheric soil and atmospheric Pb (Figure 5.2). This is the first time that the PbB seasonality patterns in a city could be explained using atmospheric Pb. Importantly, this study appears to have solved the PbB seasonality question that has been observed by other researchers for many years in the United States (Table 5.2), however the drivers of this seasonality had not been fully explained. Previously, Laidlaw et al. (2005) was able to predict indirectly seasonal variations in children's PbB levels in Indianapolis, Indiana New Orleans, Louisiana and Syracuse New York by the use of soil re-suspension variables as independent predictors. However, atmospheric Pb data was not available to model at the time. Havlena et al. (2009) were able to predict seasonal variation in Wisconsin Children's PbB using particulate matter less than 2.5 µm ($PM_{2.5}$). However, atmospheric Pb has never been previously used to successfully predict children's blood Pb levels following the elimination of Pb in petrol.

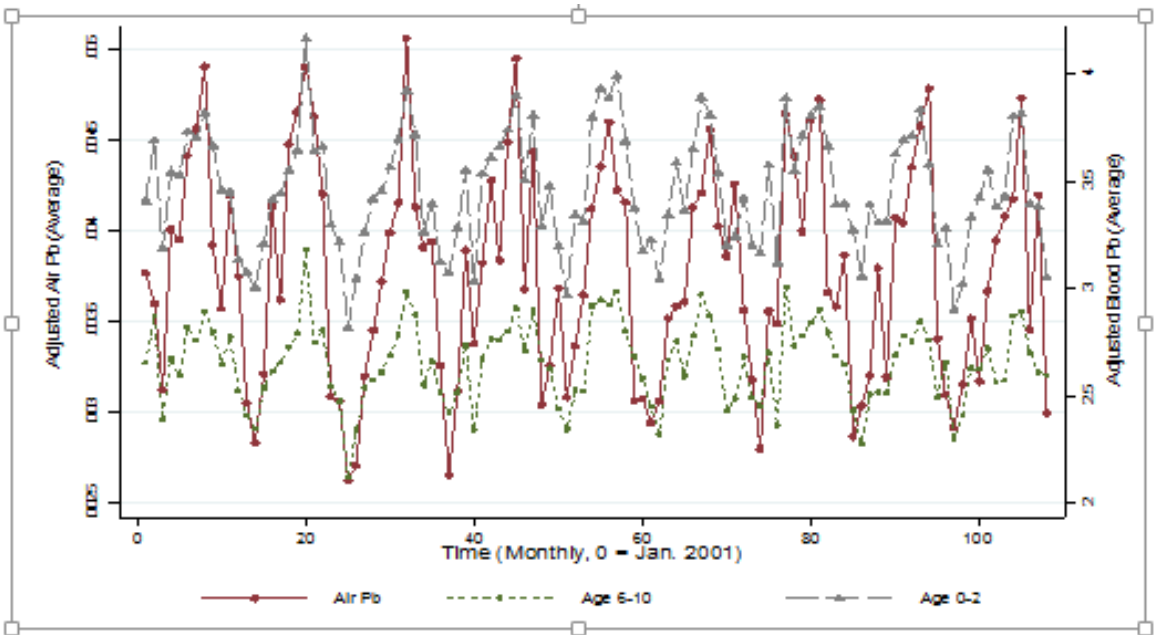

Figure 5.2 - Weather-adjusted air Pb (µg/m^3) and blood Pb (µg/dL) by age group. Average monthly child blood Pb levels adjusted by local weather conditions, child gender, method of blood draw, and census tract fixed effects (Zahran et al., 2013a; Chapter 4).

Area	Reference
Chicago, Illinois (USA)	Blanksma et al., 1969
Birmingham, England (UK)	Betts et al., 1973
Lansing, Michigan (USA)	Hunter, 1978
New York, New York (USA)	Billick et al, 1979
Connecticut (USA)	Stark et al., 1980
Boston, Massachusetts (USA)	USEPA, 1995
Los Angeles, California (USA)	Rothenberg et al., 1996
Milwaukee, Wisconsin (USA)	USEPA, 1996
Syracuse, New York (USA)	Johnson et al., 1996
Jersey City, New Jersey (USA)	Yiin et al., 2000
New York State (USA)	Haley and Talbot, 2004
Indianapolis, Indiana; Chicago, Illinois; New Orleans, Louisiana (USA)	Laidlaw et al., 2005
Milwaukee, Wisconsin (USA)	Havlena et al., (2009)
Detroit, Michigan (USA)	Zahran et al., (2013)

Table 5.2 – List of Blood Lead Seasonality Studies

5.1.4 Review of Australian Soil Pb and Blood Pb Studies

In the Australian context, the Laidlaw and Taylor (2011) *Environmental Pollution* study summarised the literature about soil Pb and children's PbB levels in Australia (Chapter 2). This paper concluded that soil is highly contaminated with Pb in many UAs and could potentially be a major source of exposure (Rouillon et al., 2013). This study is a significant contribution to the literature in Australia and has not been undertaken before. Using the established dose-response relationships between soil Pb and PbB (Bickel, 2010; Zahran et al.,

2011) and the published soil Pb concentrations observed in cities such as Sydney (Birch et al., 2011) this paper suggests that there remains a significant, but unconfirmed (due to a lack of PbB data) health risk to urban children exposed to Pb. Furthermore, this study demonstrated there remained a need for PbB monitoring to determine if there was a health risk to urban children. Around 10,000 PbB tests are carried out each year in Australia, however the data has not been pooled in a single database with relevant geographic information to decipher the locations most at risk.

5.1.5 Seasonal Pb Loading Pattern – Sydney, Australia

In the second *Environmental Pollution* study (Laidlaw et al., 2014a; Chapter 2) we have shown that in Sydney, Australia, atmospheric Pb loading rates display a seasonal pattern with maxima in the southern hemisphere summer and autumn, similar to the findings in North America (Laidlaw et al., 2012). This is a new finding. In addition it was observed that atmospheric Pb loading rates are associated with soil Pb levels at the house level and show a pronounced summertime effect with soils being re-suspended at higher rates during the summer. This finding of an association between atmospheric Pb loading rates and soil Pb concentration at the house level has not been reported previously in the international literature.

The question of whether child PbB seasonality exists in Sydney or Australian cities could not be answered in the Laidlaw et al. (2014a) study as large, seasonally derived datasets of children's PbB levels do not exist in Australia. Two small datasets (n = 7 children - Gulson et al., 2000 and n = 37 children - Gulson et al., 2008) have been analysed in Sydney, however they did not observe summertime maxima in child PbB. By comparison, the Detroit, Michigan child PbB dataset consisted of PbB measurements of 367,839 children (Zahran et al., 2013a). These PbBs exhibited strong seasonality, with maxima in the summer and

autumn. This high sample size permitted detection of seasonal patterns of PbB seasonality in Detroit children.

5.1.6 Spatial Association between Soil Pb and Children's Blood Pb Levels

A major purpose of the second *Environment International* (Zahran et al., 2013b; Chapter 4) study was to evaluate soil sample locations that explained the highest variation (R^2) in children's PbB levels. This was done to determine if soil sampling locations could be minimised in future soil Pb mapping exercises. In the Zahran et al., (2013b) study, two extensive data sets were combined: (i) 5,467 surface soil samples collected from 286 census tracts; (ii) geo-referenced PbB data for 55,551 children in metropolitan New Orleans, USA. The results indicated that soil Pb levels are spatially associated with children's PbB levels in New Orleans. Seventy-seven percent of the variation in children's PbB was explained by the four independent soil Pb sample location variables – house-side, residential street-side, busy street-side and open-space. This finding has developed our understanding that the source of Pb which poses the greatest risk to children is petrol because the three of the four different soil Pb location types - residential street-side, busy street-side and open-space -, are generally those where Pb in soil is sourced from petrol Pb, and were used successfully as independent variables in a model to predict children's PbB levels. Importantly, this study adds to a very small number of large sample studies that have examined the spatial relationship between soil Pb and PbB (Mielke et al., 1997; Bickel, 2010). Furthermore, the Zahran et al., (2013b) paper also concluded that residential street samples explained the highest variation in children's PbB levels and could be used solely or in conjunction with one more location type to save money in future soil Pb mapping studies. If it is assumed that the "house-side" soil samples (of homes with exterior Pb paint) contain a mixture of Pb paint and petrol-derived Pb (cf. Linton et al., 1980), it can generally be assumed that the Pb in the other sample types – residential street, busy street and open space, originates from petrol-derived sources, at least

in large older inner city areas. The four soil sample location types (house-side, residential street-side, busy street-side and open-space) explain 77% of the variation in children's PbB levels. Zahran et al., (2013b) decomposed the 77% variation of the four sample location types and observed the following source contributions to PbB: residential street (39.07%), open space (20.25%), busy street (21.97%) and foundation (18.71%). Summing the explained variance from three of the four sources where petrol Pb is likely to contribute the Pb suggests that roughly 81.29% of the decomposed variation in children's PbB levels was explained by soil Pb exposure sourced from petrol.

5.1.7 Source Identification of House Dust – Inner West of Sydney

The principal aim of the *Environmental Pollution* study (Laidlaw et al., 2014a; Chapter 2), was to determine the predominant source(s) of Pb inside a typical western Sydney brick homes. Using Pb isotopic composition and XAS, it was concluded that petrol (gasoline) derived soil Pb was the dominant source of Pb in house dust in the interior of the western Sydney homes (Laidlaw et al., 2014a). This is the only known study to analyse simultaneously Pb isotopic composition and Pb speciation using XAS for the purpose of determining the source identification of Pb in house dust. Other studies have used XAS to understand house dust soil sources (MacLean et al., 2011), but none have applied both methods to elucidate exposure sources. The agreement of the analysis from the two methods has bolstered the conclusions drawn from other data. Gulson et al., (2013) used Pb isotopes and concluded that Pb from soils was a major source of Pb in house dust. Using Australia data from Donovan (1996), Gulson et al. (2013) measured Pb isotopic and Pb concentration measurements from children's blood, floor dust wipes, soil, drinking water and paint from 24 dwellings where children had previously recorded PbB levels ≥15 µg/dL in an attempt to determine the source(s) of their elevated PbB. Results indicated that there was a strong

isotopic correlation of soils and house dust (r=0.53, 95% CI 0.20–0.75) indicative of a common source(s) for Pb in soil and house dust. XAS and Pb isotopes (Chapter 2) from the Laidlaw et al. (2014) study indicated that the predominant source of Pb in the western Sydney homes is from Pb contaminated soil with some contributions from Pb-based paints. These findings are important as the common perception is that Pb paint is the main source of Pb in house dusts (Jacobs, 1995).

The Laidlaw et al. (2014a) study found 5 lines of evidence that Pb in soil dust is a major source of Pb in house dust:

1. The XAS Pb speciation patterns in soil matched the Pb speciation patterns in house dust in two of the three Sydney homes, and partially matched in the third home, suggesting that the Pb residing in the soil is the same source of Pb in interior house dust.
2. The Pb isotope ratios of the house *dust* samples plotted near the house *soil* samples and near the petrol isotope Pb ratios and plotted away from the Pb paint samples. This suggests that the Pb in the house dust originates from Pb in soil and is ultimately derived from Pb in petrol.

3. Exterior soil Pb concentrations and interior vacuum dust Pb concentrations were significantly correlated (r = 0.659, p < 0.001). Each 1 mg/kg increase in soil Pb induces a 0.803 mg/kg (95 % CI, 0.369 to 1.238) increase in the median of the distribution of Pb content of vacuum dust. These homes did not contain exterior Pb paint. Other studies of household Pb dust have drawn parallel conclusions. For example, in England (UK), Thornton (1990) observed a highly significant relationship

between the concentration of Pb in house dust and garden soil (r = 0.531, p = 0.001, n = 4512). Similarly, in the United States, Bornschein et al. (1986) also observed that soil Pb and house dust Pb concentration were closely correlated (R^2 = 0.57).

4. Exterior soil Pb concentrations are significantly correlated with interior petri-dish Pb loading rates ($p < 0.001$) (cf. Gulson et al., 2006a). This suggests that the Pb deposited in the petri dishes originated from re-suspended outdoor soil which has penetrated the interior of the home and deposited in the petri-dishes.

5. The correlation between exterior atmospheric Pb loading rates and interior vacuum Pb concentrations in this study is significantly positive (r = 0.314, $p < 0.001$). This is important because it suggests that there is a temporal exposure pathway between exterior Pb loading rates and interior house dust Pb concentration fluctuations. No other long-term study has demonstrated that simultaneously measured interior house dust Pb concentrations and exterior Pb loading rates are correlated across multiple houses. Gulson et al. (2006a) observed that dust-fall accumulation was the only significant predictor of children's PbB levels in his study of 113 children in Sydney.

Thus, in summary, the evidence indicates strongly that soil Pb, derived from petrol/gasoline is a major source of house dust Pb in Sydney homes.

The literature supports the hypothesis that significant proportions of Pb inside some homes originates from Pb bound to soil dust particles with some contribution from Pb paint sources. Rabinowitz (1987) analysed Pb isotopes in interior house dust of Boston homes that had never been painted with Pb based paint. It was observed that the Pb isotopes in the interior house dust matched the Pb isotope ratio in urban soils. Chemical mass balance was used to apportion

the major proximate contributors of Pb mass to house dust in 64 urban Jersey City, New Jersey, homes with Pb-based paints (Adgate et al., 1998b). Coarse (up to 60 μm) and PM_{10} (<10 μm) particle size fractions of vacuum dust samples were collected simultaneously. The Adgate et al. (1998b) study observed that crustal materials and deposited airborne particulates were responsible for approximately two-thirds of the Pb mass to house dust and interior Pb-based paint sources contributed the remaining third. Furthermore, using Pb isotope ratio analysis, Adgate et al. (1998a) found that about half of the Pb in house dust of 10 homes in Jersey City, originated from sources outside the home, such as soil. Speciation of Pb by XAS by Pingitore et al. (2010) indicated that Pb compounds associated with Pb-based paint accounted for perhaps half of the Pb in 24 household dust wipes. Soil-derived, sorbed Pb was also a major species in many of Pingitore et al.'s (2010) dust wipes, suggesting that soil remediation is required to reduce exposure risk. This finding is consistent with the 4 house Pb study of Sydney homes (Laidlaw et al., 2014a). Similarly, in Canada, MacLean et al. (2011) used XAS to determine the source of Pb in an archived vacuum bag dust sample with a Pb concentration of 1670 mg/kg of which 95% was bio-accessible. They concluded that both Pb paint and soil were the sources of the Pb in the house. In the London England borough of Richmond, Hunt et al. (1993) used automated scanning electron microscopy (SEM) in conjunction with energy dispersive X-ray spectroscopy to analyse Pb particles in 16 homes of various ages. Hunt et al. (1993) concluded that paint, road dust and garden soil are the major contributors of particulate Pb and the primary contributing source in the 64–1000-μm size range of the house dusts appeared to be paint, but in the <64 μm size fraction of the dusts paint, road dust and garden soil all make significant contributions. Grain size is important as smaller particles have higher Pb concentration (up to 5 times the concentration observed in bulk soil) and higher bio-availability, the smaller particles preferentially adheres to children's hands and are ingested via hand to mouth behaviour (Juhasz et al., 2011).

5.1.8 Establishing Petrol-derived Pb as a Major Source of Lead in Soil, House Dust and Children's Blood

The studies in this dissertation found five primary lines of evidence showing that petrol-derived Pb is a major source of Pb in urban inner-city soil, house dust and children's PbB:

1) The *Environment International* (Mielke et al. 2011) and *Science of the Total Environment* (Mielke et al. 2010) papers both showed that an enormous volume of petrol-derived Pb has been emitted into inner-city environments in the US and is now residing in urban soils which are being re-suspended into the atmosphere (*Atmospheric Environment*; Laidlaw et al., 2012);
2) The Laidlaw et al., (2014a) *Environmental Pollution* paper observed that the Pb isotopic composition ratios of house dust clustered near the petrol Pb isotope signature;
3) The Zahran et al., (2013a) *Environmental Science and Technology* paper showed clearly that re-suspended atmospheric soil and atmospheric Pb were associated with children's PbB levels. The Laidlaw et al. (2012) *Atmospheric Environment* paper showed that atmospheric soil and atmospheric Pb correlated and that atmospheric soil and atmospheric Pb were three times higher on weekdays compared to weekends, suggesting that turbulence impacting on Pb contaminated road-side soils sourced from petrol is the source of seasonal variations in children's Pb poisoning;
4) The Zahran et al. (2013b) *Environment International* paper demonstrated that 77% of the spatial variation in children's PbB levels was associated with four different soil Pb location types – house-side, residential-street, busy-street and open area. These location types were used as independent variables in a model to predict children's PbB levels. Three of these soil Pb sampling location 'types' are associated with petrol-derived Pb - residential-street, busy-street and open area, while the foundation soil

sample type is thought to contain a mixture of exterior paint and petrol-derived Pb (Linton et al., 1980); and

5) The exterior of the Sydney homes and neighbouring homes in the Laidlaw et al., (2014a) *Environmental Pollution* paper did not contain Pb paint. This further supports the contention that the Pb in the soils of the homes (and the interior of the homes) originated from a petrol (gasoline) source.

Collectively, the evidence suggests that not only is soil Pb a major source of Pb poisoning in children, but the source of a large portion of the soil Pb in some areas with high soil Pb concentrations in large inner-city areas is derived from past use of Pb in petrol. This however, does not mean that there are no significant contributions from deteriorating exterior Pb-rich paints which may predominate adjacent to homes with exterior Pb paint and in smaller cities (Clark and Knudsen, 2013). The studies show clearly that Pb from petrol, is a major source of contamination that has not been given equal weight in the attribution of exposures sources of Pb in young children.

5.2 LIMITATIONS

In the absence of smelter sources in UAs, the two most common sources of Pb in urban soils are from petrol (gasoline) and exterior Pb-based paint. It is suggested that in large inner cities such as New Orleans and Sydney, the predominant source of Pb is from petrol (Mielke et al., 2010; Laidlaw et al., 2014a), while the house-side soils in homes with Pb-based paints in these cities contains a mixture of Pb from petrol and Pb-based paints (Linton et al., 1980). However, in older areas of smaller cities it is suggested that Pb paint may be the major source of Pb in soils due to a lack of historical traffic volume compared to large cities (Clark and Knudsen, 2013). Caution should be made when inferring the findings of the Laidlaw et al.

(2014a) *Environmental Pollution* Paper to other homes as it is important to consider site specific sources, which may be dependent on the presence and condition of Pb-based paints and whether Pb paint remediation has been performed. Also, causality between atmospheric soil re-suspension and Pb aerosol concentrations observed in the Laidlaw et al. (2012) study was inferred and could not be proved; although it is noted that the association is strongly correlated and consistent between the cities examined. Consequently, the ensuing potential health impacts of soil Pb re-suspension as indicated by other studies can also only be inferred.

5.3 CONCEPTUAL MODEL

The following conceptual model was developed based upon the results of this dissertation. During the summer and autumn, Pb in urban roadside soils and road dusts is being re-suspended into the atmosphere primarily by the interaction between low soil moisture and traffic induced turbulence. The resulting soil re-suspension results in elevated atmospheric Pb loading rates that are proportional to the local soil Pb concentrations. This process is the current dominant source of atmospheric Pb in urban environments lacking other major sources such as Pb smelters or automotive Pb. Following re-suspension, atmospheric Pb penetrates into the interior of homes and settles on to contact surfaces where its dust concentration correlates spatially with soil Pb concentrations and temporally with exterior atmospheric Pb loading rates, with maxima in the summer and autumn. It is also tracked into home contact surfaces via shoes and pets. Unsuspecting children are then exposed primarily via hand to mouth contact as well as inhalation. Children are also exposed directly to Pb in outdoor soil via hand to mouth activities (thumb sucking and ingestion), particularly in the summer in the northern hemisphere. Subsequently, taken as a group, inner city children's *low level* lead poisoning will correlate strongly with the re-suspended and settled atmospheric Pb with maxima in the summer and autumn. Their PbB will also correlate spatially with soil Pb

concentrations. Children also can also be exposed to additional sources such as flaking Pb paint dust and chips and Pb contaminated drinking water, where present.

5.4 IMPLICATIONS OF FINDINGS

Government public health agencies have generally focused their entire efforts at eliminating Pb poisoning by performing Pb-based paint removal and remediation. In the US, the Centers for Disease Control and Prevention (CDC) acknowledge that along with Pb-based paint and Pb dust, soil is a major Pb source of Pb poisoning in children. However, the agency does not provide a single recommendation about soil Pb remediation in their guidelines (CDC, 2007). Australia has a soil Pb residential housing guideline of 300 mg/kg (NEPM, 2013), but there is no specific legislation requiring analysis of residential soils. Contaminated land investigations are almost entirely performed on industrial contaminated sites, not non-point legacy residential soil Pb contamination, although studies in mining and smelting towns such as Boolaroo, Mount Isa, and Port Pirie show that soil Pb remains a significant source of exposure (Willmore et al. 2006; Taylor et al., 2010; 2013; Mackay et al., 2013). Research in this dissertation suggests that the CDC and other public health agencies worldwide need to refocus their efforts primarily towards inner-city urban *residential* soil Pb exposure in order to achieve the objective of eliminating the adverse effects of low level PbB poisoning (NTP, 2013). Given that PbB seasonality patterns cannot be explained by Pb paint exposures, this study has demonstrated conclusively shown that PbB seasonality patterns are correlated with external atmospheric Pb concentrations that have originated from re-suspended urban Pb contaminated soil. There are numerous cities globally where the soil Pb concentrations are highly contaminated due to past emissions of Pb in gasoline and where these processes are likely to be occurring (Laidlaw, 2014).

Systematic PbB poisoning screening is needed in Australian inner-cities where soils are highly impacted by Pb. Taylor et al. (2012, 2013) estimates that up to 100,000 Australian children may have PbB levels at concentrations that could be detrimental to children's health.

Furthermore, studies in this thesis indicate strongly that Pb contaminated urban soil is also implicated as the major source for atmospheric Pb aerosol loadings. A consequence of the re-suspension of Pb contaminated soil is that it has significant repercussions for ongoing adverse Pb exposures in urban children. Pingitore et al. (2009) observed that if Pb contaminated urban soil is the principal source for airborne Pb in urban settings, then "*contaminated soil may set a practical lower limit for future decreases in regulation of airborne Pb levels*" (Pingitore et al., 2009, p. 5).

5.5 SOIL PB RISK MANAGEMENT OPTIONS

To reduce the Pb exposure to meet lower guidelines, large US urbanized areas will probably require extensive environmental treatment, and soil Pb is one factor that can be modified to reduce children's Pb burdens (Filippelli and Laidlaw, 2010). Fortunately, as described, previously clean soil with a median Pb content of 16.5 mg/kg is available nearby all US urbanized areas (Gustavsson et al., 2001). Similarly low Pb soil is available in Australia. The median and mean soil Pb background concentrations in the Sydney region are around 15.5 and 23.3 mg/kg, respectively (Olszowy et al., 1995).

In Minneapolis, Minnesota a pilot project was conducted that successfully reduced children's exposure and prevented the expected seasonal summertime PbB increases (Mielke et al., 1992). The method was refined in New Orleans, Louisiana where abundant and remarkably clean (median ~5 mg/kg Pb) Mississippi River sediments are available as a natural resource for covering Pb contaminated urban soils (Mielke, 2005; Mielke et al., 2006a,b). Other pilot

projects focused on soil Pb were conducted in Boston, Massachusetts and Chicago, Illinois with varying success (US EPA, 2001; Binns et al., 2004).

A full-scale national program is underway in Norway to clean up contaminated soils at all childcare centres, elementary schools and parks in the ten largest cities (Ottesen et al., 2008). The differences between the US approach and Norway's program are striking: in the US secondary prevention is conducted after a child is identified with elevated PbB. In Norway, the World Health Organisations (WHO) principles of the precautionary principle are followed (Louis et al., 2006). The Precautionary Principle is defined by UNESCO (2005) as the following: *When human activities may lead to morally unacceptable harm that is scientifically plausible but uncertain, actions shall be taken to avoid or diminish that harm. Morally unacceptable harm refers to harm to humans or the environment that is*

- *threatening to human life or health, or*
- *serious and effectively irreversible, or*
- *inequitable to present or future generations, or*
- *imposed without adequate consideration of the human rights of those affected.*

Actions are interventions that are undertaken before harm occurs that seek to avoid or diminish the harm. Actions should be chosen that are proportional to the seriousness of the potential harm, with consideration of their positive and negative consequences, and with an assessment of the moral implications of both action and inaction. Therefore, the emphasis is on primary prevention whereby environmental contaminants are directly addressed and treated; children's PbB samples are not included as part of the protocol. In the US the current residential soil guideline is 400 mg/kg Pb for the bare soil of a play area, and 1200 mg/kg for the remaining areas of the property (ATDSR, 2013). By contrast, in Norway the soil Pb guideline for play areas is 90 mg/kg (Environment Norway, 2013), and there is discussion about reducing the guideline to 60 mg/kg. In Australia the residential soil Pb guideline was reaffirmed in 2013 at 300 mg/kg (NEPM, 2013).

Research in New Orleans, Louisiana indicates that communities with a median soil Pb ≤80 mg/kg would generally prevent children from reaching a PbB ≥10 μg/dL (Mielke et al., 1999). If a stricter PbB guideline is observed that reflects the current clinical health effects of Pb, then the soil Pb guideline must also be significantly lower. Many US urbanized areas have soil Pb medians above 100 mg/kg (Mielke, Laidlaw et al., 2011). Norway's precedence for implementing a national clean play area program serves as a model for commitment to primary prevention that benefits children and ultimately all community members of Pb impacted urbanized areas (Ottesen et al., 2008). In contrast to the US EPA's soil Pb guideline, the recently revised Californian soil Pb guideline level is set at 80 mg/kg in order to keep PbB below 1 μg/dL, which was deemed to be less than one IQ impact (OEHHA, 2009).

In New South Wales (NSW), section 149 Planning Certificates are issued in accordance with the *Environmental Planning & Assessment Act 1979* (NSW). The certificates contain information on how a property may be used and the restrictions on development. When land is bought or sold the *Conveyancing Act 1919* (NSW) requires that a Section 149 Planning Certificate be attached to the Contract for Sale. Under sections 149 (5) and (6) of the *Environmental Planning & Assessment Act 1979* (NSW) there is a clear opportunity to identify actual or potential contamination at properties:

> *(5) A council may, in a planning certificate, include advice on such other relevant matters affecting the land of which it may be aware.*
>
> *(6) A council shall not incur any liability in respect of any advice provided in good faith pursuant to subsection (5). However, this subsection does not apply to advice provided in relation to contaminated land (including the likelihood of land being*

contaminated land) or to the nature or extent of contamination of land within the meaning of Part 7A.

Therefore, following proper environmental assessment of potentially contaminated areas and properties for dust or soil Pb issues, a section 149 certificate should include the assessment information so that prospective purchasers are aware of any potential risk to health.

There are multiple international soil Pb risk management practices currently being utilized, which are summarised as follows:

- Excavation in soil Pb contaminated areas involves the removal of Pb contaminated surface soil and disposal at a landfill. This is considered to be the gold standard approach, however it is very expensive.
- Interrupting children's summer and autumn PbB poisoning patterns requires interrupting the soil Pb pathway by soil excavation, soil covering or maintaining soil moisture. Remediation of roadside (0-25m or 0-50m from the roadside) soils may be required to interrupt exposure to re-suspended roadside soils and requires further research. Given that roadway soil re-suspension appears to be a major source of Pb dust in the urban atmospheric environment, it is recommended that the top 15 to 20 cm of Pb contaminated surface soil be excavated along major thoroughfares in the urban environment. Alternatively, this exposure pathway could be by interrupted by covering the soil with a geo-textile fabric and then covering it with clean soil and growing a dense lush grass cover. Soil covering describes the process by which Pb contaminated surface soils are isolated from contact typically via covering the existing soil with a geo-textile fabric followed by covering the geo-textile with 10-15 cm of clean topsoil and then growing grass in the soil or placing sod on top of the soil.

- In Australia, precedent for the remediation/isolation of urban soils has been set in Boolaroo NSW near the location of the former Pasminco smelter. The NSW Department of Environment and Climate has approved a soil Pb abatement strategy for approximately 4000 properties surrounding the former Pasminco smelter at Boolaroo (Lake Macquarie Council, 2009). Soils with Pb concentration ranging between 300 mg/kg and 2,500 mg/kg will be covered with clean soil and in areas where soil concentrations exceed 2,500 mg/kg the top 5 cm of soil will be removed.
- In the US, Mielke et al. (2006a,b) demonstrated that exposure to Pb contaminated urban soils can be prevented by covering contaminated soils with about 15 cm of low Pb (median ~5 mg/kg) soil. To achieve that, clean soil is simply graded over the old soil layer, hydro-seeded (a slurry of seeds and moisture-retaining fill mixture sprayed onto the ground), and left to grow into a lawn. This approach "caps" the Pb-contaminated soils, and prevents children from coming into contact with soil-borne metals. Yard remediation has been demonstrated to effectively reduce PbB levels. For example, Maisonet et al. (1997) found that yard soil remediation was a protective factor for elevated PbB levels in children (odds ratio, 0.28; confidence interval, 0.08-0.92).
- The re-suspension of Pb contaminated soil can also be limited by maintaining high soil moisture. However this method may not be effective in Australia given the scarcity of water and does not prevent direct exposure. Soil moisture can be maintained during periods of high evapo-transpiration which can reduce re-suspension of soils. This 'wetting or watering down technique is commonly used at mine sites.

- Park et al. (2011) concluded that application of phosphate solubilising bacteria with locally available low-cost insoluble phosphate compounds can be used as a sustainable remediation technology for *in situ* stabilization of Pb in urban soils contaminated by Pb. However, there may be a risk of the release of phosphorous to the environment utilizing this strategy. It requires further evaluation.
- Soil Pb concentrations can be diluted by applying clean topsoil through the grass cover and allowing the grass to grow through the new soil. The effectiveness of this technique has not been evaluated scientifically.
- Effort could also be made to ensure that homes are well sealed to prevent the migration of dust into the home – tricky in older homes. This includes making sure that all migration pathways into the home are well sealed. During periods of high evapo-transpiration, in areas where Pb in soil is contaminated, residents may reduce exposure by operating an air conditioner and making sure the air filter is not clogged.
- Phytoremediation techniques utilize the preferential uptake of Pb in plants for remediating Pb in soils. However, this technique is considered ineffective and impractical for large scale remediation that is required in inner-city soils.
- Residents in soil Pb contaminated areas should also remove shoes prior to entering the home and should not bring soiled clothes into the home. Soiled clothes should be washed outside and pets' feet and fur should be cleaned or brushed prior to coming inside.

Some Pb exposure reduction tips that were compiled during this PhD study are presented in Appendix B.

5.6 SCOPE FOR FUTURE WORK

The following work is recommended:

- It is recommended on the basis of the findings presented in this thesis that the Australian and US governments should commission high density soil Pb geochemical maps (sample depth = 0-2 cm) across all the major cities to better understand where the general soil Pb exposure risks are located. These maps should be published online in one central location for each government and a press release should be provided to all major newspapers upon completion. However, these maps must not be considered to represent the soil Pb concentrations at individual homes. Site specific sampling is required to assess the risk at children's homes.
- In homes residing in urban inner city areas it is recommended that three soil samples be collected and analysed for Pb in each home with children under the age of 7. One sample should be collected in the middle of the front yard, the second sample in the middle of the back yard and the third soil sample collected 1 m beside the foundation at the front of the house. A vacuum bag dust sample (best if collected in the summer) should be collected as well. The soil samples should be sieved to less than 150 µm grain size. The results from the analyses of the samples could be entered into an IEUBK model (USEPA, 2013) to predict PbB levels. Alternatively, similar PbB models applying empirically derived equations relating soil Pb and PbB concentration could be utilized (Bickel, 2010; Zahran et al., 2011). This process is preferable to one which requires blood to be drawn to determine PbB level. It would allow a parent to predict the risk associated with the levels of Pb at their residence and the risk of Pb poisoning, independent of Pb paint exposure.
- An evidenced based review and assessment of various soil Pb remedial techniques with cost-benefit analysis of each technique should be conducted and published in the peer-reviewed literature to help direct council, governments, EPAs health etc.

CHAPTER 6

Conclusions

This thesis has applied an evidence-based approach to the analysis of Pb in paint, soil, dust, blood, homes and atmosphere to inform the debate about the major sources of Pb that contribute to the ongoing poisoning of children in inner-city environments. The results support the argument / hypothesis that the elevated soil Pb levels found in high population inner-city environments are sourced primarily from petrol. While this conclusion does not exclude contributions from exterior paint, it is clear that the legacy emissions from leaded petrol form a major source of Pb to which inner-city children continue to be exposed at adverse levels in both Australia and the USA. While there remain two viewpoints about the source of Pb poisoning children – "paint only" and "soil source", it is clear that both Pb in soil and Pb in interior and exterior paint can both be major contributors to children's Pb poisoning. Consequently, in trying to unravel source apportionment in different locations and situations, both of these major sources must be assessed in order to eliminate the scourge of low and high level Pb poisoning. Failure to address the soil Pb pathway (regardless of paint or petrol source) will result in a continuation of inner city low level PbB epidemics, as detailed within this study and in the broader literature.

The findings in this thesis indicate strongly that summer and autumn re-suspension of Pb contaminated roadside soils is likely to be a primary driver of the PbB seasonality phenomena observed for many years in the US. The good news arising from within this thesis is that this exposure pathway could be easily eliminated by covering roadside grass and soils (0-25 to 0-50 m from roadside) with a geo-textile fabric and covering with clean soils and growing a lush grass cover. Furthermore, it is recommended that inner-city residents who have children aged 0-7 years in large cities analyse their soil for Pb concentrations. The results from the analyses of the samples could be entered into an IEUBK model (USEPA, 2013) to predict PbB levels. Alternatively similar PbB models applying empirically derived equations relating

soil Pb and PbB concentration could be utilized (Bickel, 2010; Zahran et al., 2011). This process is preferable to one which requires blood to be drawn to determine PbB level. It would allow a parent to predict the risk associated with the levels of Pb at their residence and the risk of Pb poisoning, independent of Pb paint exposure. If predicted PbB levels exceed 5 µg/dL, then intervention may be warranted in order to lower and eliminate future exposures.

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APPENDIX A

Pb Dust Exposure Prevention Tips

The following Pb exposure prevention tips are suggested if Pb is a concern in the environment:

- Remove shoes at the door and use a commercial-quality door mat to reduce track-in. Feet should be wiped at least twice on the door mat.
- Purchase a High-Efficiency Particulate Air (HEPA) filter vacuum if possible.
- Vacuum rugs, mats and carpets and damp mop floors once a week (twice a week if there are toddlers in the home). Vacuum each section of carpet at least four times.
- Do not vacuum hard surfaces.
- Clean window frames, windowsills, and other surfaces weekly. Use a mop or sponge with warm water and a general all-purpose cleaner or a cleaner made specifically for Pb.
- Thoroughly rinse sponges and mop heads after cleaning dirty or dusty areas.
- Inspect the vacuum belt and bag monthly. Change bag when it is half full.
- Cleaning surfaces below five feet that small children may put their mouths on, including but not limited to wall corners, doors, stairs, railings, windows, baseboards, and chair rails and parts of windows (with sills below five feet) that move or touch moving parts (once a month).
- Keep toddlers away from remodelling areas, peeling paint, foundation soil, and window wells.
- Keep play areas clean. Wash bottles, pacifiers, toys, and stuffed animals regularly.
- Lay down a clean sheet before putting a baby on a carpet.
- Keep children clean. Ensure that children wash before they eat. Wash toddler's hands often.
- Protect food from settled dust by covering and/or washing.
- When remodelling, seal off the work area and avoid tracking dust from remodelled area.
- Cover bare soil in gardens and areas beside house with wood chips or mulch.
- Grow grass where soil is bare, or place wood chips or mulch on bare soil.

- Make sure children eat regular nutritious meals, since more Pb is absorbed on an empty stomach. Make sure children's diets contain plenty of iron and calcium: Examples of foods high in iron are liver, fortified cereal, cooked beans, spinach, and raisins. Examples of foods high in calcium are milk, yogurt, cheese, and cooked greens.

- Have children wash their hands after playing outside, and before meals, naps and at bedtime.

- *Regularly wash children's toys.* Toys can become contaminated from household dust or exterior soil.

- Keep pets outside. If allowed inside, brush fur outside and clean all dirt from paws.

- Clean up paint chips immediately.

- *Prevent children from playing in bare soil; if possible, provide them with sandboxes***.** If using a sandbox, parents should also cover the box when not in use.

- Do not remove Pb-based paint yourself. It is extremely hazardous.

- If soils are contaminated, avoid growing leafy and root vegetables.

- If soils are contaminated, wear gloves when handling soil and wash gardening clothes separately from regular wash. Avoid bringing soil into the house.

In the event that a resident has a high PbB level, a more aggressive approach may be warranted, which may include: removing ceiling dust, removing or covering contaminated soil around the home, stabilising flaking Pb-based paints, cleaning or replacing carpets and cleaning soft furnishings.

- To permanently remove Pb hazards, you must hire a certified Pb "abatement" contractor. Abatement (or permanent hazard elimination) methods include removing, sealing, or enclosing Pb-based paint with special materials.
- Always hire a person with special training for correcting Pb problems—someone who knows how to do this work safely and has the proper equipment to clean up thoroughly. Certified contractors will employ qualified workers and follow strict safety rules set by their state or the federal government.

APPENDIX B

Australian Synchrotron Article

BEAMLINES

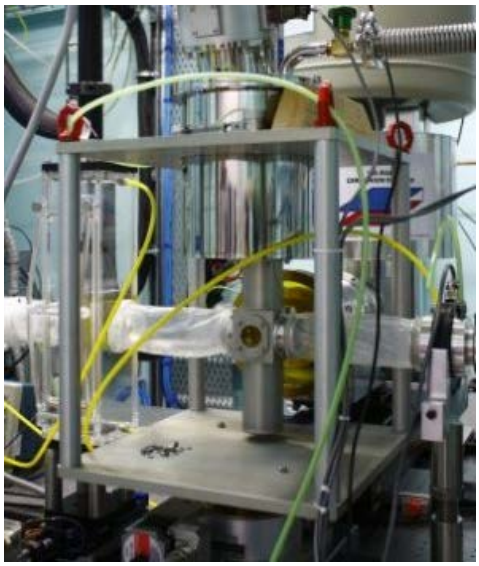
CITY SOILS PROVIDE NEW LEAD

The main source of high lead levels found in homes in western Sydney is not interior lead paint but lead from soils, according to a Macquarie University research investigation. Garden soils have accumulated lead mainly as a result of decades of emissions from vehicles running on leaded petrol. Other sources of lead include the deterioration of exterior leaded paint.

The preliminary findings were made by Mark Laidlaw and Mark Taylor from Macquarie University, whose international collaborators include researchers at Colorado State University and the University of Texas at El Paso. Over a 15-month period, Mark and Mark collected data from paint samples, surface and subsurface soils, interior dust collected by vacuum cleaning, settled interior and attic dust, and exterior atmospheric dust obtained from four inner western Sydney houses and one in an area of low density bushland 28 kilometres northwest of Sydney. None of the houses had exterior paint, except on gutters and windows.

Mark and Mark analysed their samples for total metal content (arsenic, cadmium, chromium, copper, lead, mercury, nickel and zinc) and lead isotopes, and used x-ray absorption spectroscopy at the Australian Synchrotron to obtain lead spectra from each sample. The researchers then used the distinctive lead spectra, which vary according to the different lead compounds present in the samples, rather like fingerprints to determine where the lead inside the homes had come from.

Right: sample chambers in the experimental hutch for the XAS beamline



The Sydney findings are consistent with earlier studies by US researchers, who found that soil accounts for around two-thirds of the lead in house dust with interior lead-based paint sources contributing the remainder. A separate UK study found that the proportion of road dust and garden soil was higher in the smaller particles of house dust that were more likely to cling to children's hands.

How does lead from outside soil and dust get into the house? "It's usually carried inside on the soles of dirty footwear or by pets," Mark Laidlaw explains. "It can also drift or be blown inside houses in the form of tiny airborne particles."

Mark and Mark also found that exterior atmospheric lead loading rates and interior vacuum bag lead levels were lower in winter than in summer.

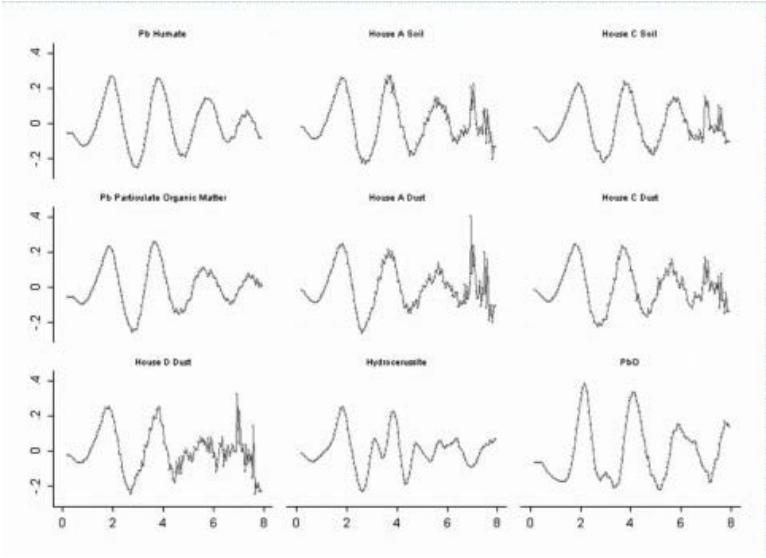
Increased lead exposure is known to adversely affect intelligence levels in children. High exposure levels in early childhood may also increase the risk of ADHD, aggression and delinquency, reduce dental health and delay sexual maturation. Adults exposed to high lead levels may be at risk of increased cardiovascular, reproduction and central nervous system problems.

In the US, blood lead screening has revealed the existence of childhood lead poisoning in all major urban inner-city areas. Australia does not have a comparable program for collecting childhood blood lead measurements. Neither country has yet developed a strategy for dealing with lead sources that are diffuse rather than those coming from a single point such as an industrial smelter or mine site.

The researchers involved in the Sydney study concluded that soil and household dust remediation should be the primary strategy for cleaning urban areas where children were presenting with blood lead poisoning and soil lead concentrations were highly elevated. The same remediation strategy could also be applied to areas contaminated by flaking lead paint, along with professional removal and sealing of any flaking lead paint.

"Identifying at-risk areas would require widespread assessment of lead levels in children's blood and in surface soils." This work should focus on the older parts of large Australian cities," Mark Laidlaw says. "This task would be best carried out by government or, alternatively, by researchers interested in understanding lead in soil and exposure risks."

Figure 1 (immediately below) shows lead spectra for houses A and C soils, houses A, C and D vacuum bag dust, lead humate, lead particulate organic matter, and typical lead paint compounds hydrocerrusite and lead oxide (PbO). Spectra for lead humate, house A soil and dust, house C soil and dust, and lead particulate organic matter are all similar, and quite different from paint hydrocerussite and PbO. House D dust is also similar to dust from A and C. Lead humate forms exclusively in soils. Image: Mark Laidlaw, Macquarie University



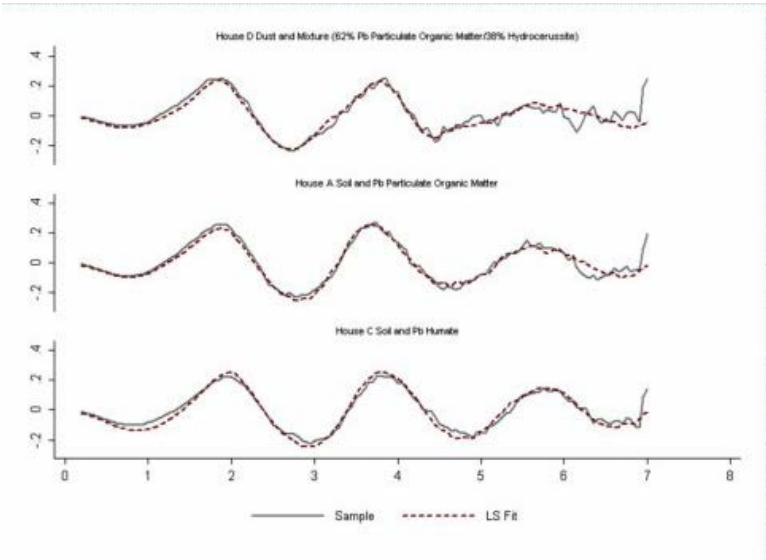
Labels

Top row L-R: Pb humate, House A soil, House C soil

Middle row L-R: Pb particulate organic matter, House A dust, House C dust

Bottom row L-R: House D dust, hydrocerussite, PbO

Figure 2 (below) shows that the lead spectrum for house D fits well with the spectrum for a mixture of lead particulate organic matter (62%) and hydrocerussite (38%). There is also a good fit between house A soil and particulate organic matter, and between house C soil and lead humate.



Labels

Top row: House D dust and mixture (62% Pb particulate organic matter / 38% hydrocerussite)

Middle row: House A soil and Pb particulate organic matter

Bottom row: House C soil and Pb humate